VOLUME 69 • MAY 1959 • NUMBER 5

A.M.A. ARCHIVES OF OTOLARYNGOLOGY

Principles Used in the Development of Modern Temporal Bone Surgery
Julius Lempert

Functional Microsurgical Procedure in Middle Ear for Chronic Purulent Otitis
Enrico Bozzi

Some Observations on the Pathogenesis of Middle-Ear Cholesteatoma
César Fernández, John R. Lindsay, and Merle Moskowitz

The Value of Self-Inflation of the Middle Ear
Aubrey G. Rawlins

The Localization of Acetylcholinesterase in the Cochlea
Harold F. Schuknecht, John A. Churchill, and Rosemary Doran

Congenital Hereditary Kidney Disease and Hearing Loss
Robert E. Klotz

The Electrolabyrinthogram
M. Spector, E. G. Szekely, and E. A. Spiegel

Postcricoid Carcinoma and Obstructing Lesions of the Thoracic Esophagus
Henry J. Heimlich

Pathology and Therapy of the Singing Voice
Paul J. Moses

Neurilemmoma of the Hypoglossal Nerve
Claude D. Winborn, Daniel M. Martinez, Bruce F. Holding Jr., and James B. Hutcheson

Laryngofissure: Indications and Technique
John C. Lillie and Kenneth D. Devine

Pharyngeal-Esophageal Diverticulum
José A. Arruda Botelho

A Comparative Study of Nasal Decongestion by Sympathomimetic Drugs
Domingo M. Aviado Jr., A. L. Wnuck, and Edwin S. de Beer

The Efficiency of Limited Frequency Audiometric Screening in a School Hearing Conservation Program
Clifton F. Lawrence and Wallace Rubin

Use of the Plastic Needle in Transtracheal Anesthesia
Heston L. Wilson

A Bilateral Posterior Intranasal Pack
Sidney H. Gidoll

New Instrument—Roller Knives
Harold F. Schuknecht

Bronchoesophagology
F. Johnson Putney

Abstracts from Current Literature

News and Comment

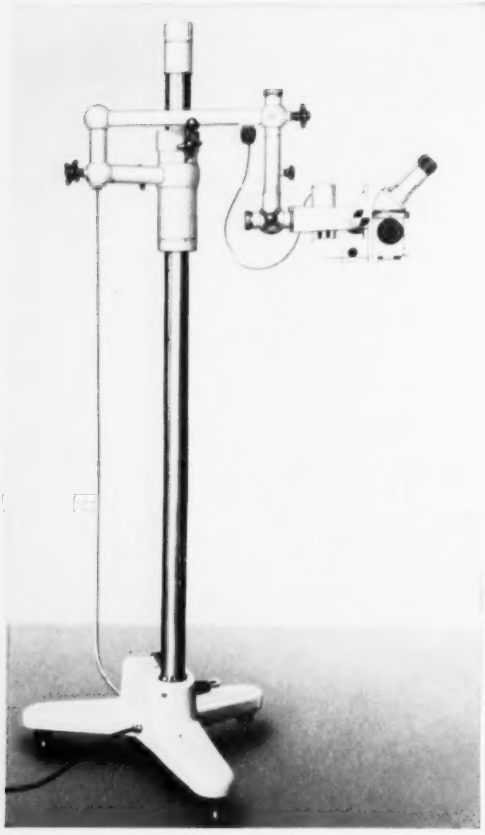
Directory of Otolaryngological Societies

AMERICAN MEDICAL ASSOCIATION PUBLICATION

NOW AVAILABLE

The Mueller Zeiss Operating Microscope with

NEW EXPLOSION-PROOF FOOTSWITCH



Only from V. Mueller— the Shambaugh-Derlacki Model with sterilizable stainless shields for eyepieces, objectives.

Now you can order the Mueller Zeiss Operating Microscope with an explosion-proof footswitch. The scope is modified in V. Mueller shops to enclose completely the transformer and base assembly; and the lower electrical connection is locked in place, making it nonremovable, therefore nonsparking.

This safety modification can be made on scopes now in use, if returned to our shops. Write for estimate.

Ask for literature on our complete line of stapes mobilization instruments.

V MUELLER & CO.

Fine Surgical Instruments and Hospital Equipment

330 South Honore Street, Chicago 12, Illinois

Dallas • Houston • Los Angeles • Rochester, Minn.

Armamentarium

TABLE OF CONTENTS

VOLUME 69 MAY 1959 NUMBER 5

ORIGINAL ARTICLES

PAGE

Principles Used in the Development of Modern Temporal Bone Surgery
Julius Lempert, M.D., New York 515

Functional Microsurgical Procedure in Middle Ear for Chronic Purulent Otitis
Enrico Bozzi, M.D., Milan, Italy 529

Some Observations on the Pathogenesis of Middle-Ear Cholesteatoma
César Fernández, M.D.; John R. Lindsay, M.D., and Merle Moskowitz, M.D., Chicago 537

The Value of Self-Inflation of the Middle Ear
Aubrey G. Rawlins, M.D., San Francisco 547

The Localization of Acetylcholinesterase in the Cochlea
Harold F. Schuknecht, M.D.; John A. Churchill, M.D., and Rosemary Doran, B.S., Detroit 549

Congenital Hereditary Kidney Disease and Hearing Loss
Robert E. Klotz, M.D., Boston 560

The Electrolabyrinthogram
M. Spector, M.D.; E. G. Szekely, Ph.D., and E. A. Spiegel, M.D., Philadelphia 563

Postcricoid Carcinoma and Obstructing Lesions of the Thoracic Esophagus
Henry J. Heimlich, M.D., New Rochelle, N. Y. 570

Pathology and Therapy of the Singing Voice
Paul J. Moses, M.D., San Francisco 577

Neurilemmoma of the Hypoglossal Nerve
Claude D. Winborn, M.D.; Daniel M. Martinez, M.D.; Bruce F. Holding Jr., M.D., and James B. Hutcheson, M.D., Dallas, Texas 583

Laryngofissure: Indications and Technique
John C. Lillie, M.D., and Kenneth D. Devine, M.D., Rochester, Minn. 589

Pharyngeal-Esophageal Diverticulum
José A. Arruda Botelho, M.D., São Paulo, Brazil 594

A Comparative Study of Nasal Decongestion by Sympathomimetic Drugs
Domingo M. Aviado Jr., M.D., Philadelphia; A. L. Wnuck, B.S., and Edwin S. de Beer, Ph.D., Tuckahoe, N. Y. 598

AUDIOLOGY SECTION

The Efficiency of Limited Frequency Audiometric Screening in a School Hearing Conservation Program
Clifton F. Lawrence, M.A., and Wallace Rubin, M.D., New Orleans 606

CLINICAL NOTES, NEW INSTRUMENTS AND TECHNIQUES

Use of the Plastic Needle in Transtracheal Anesthesia
Heston L. Wilson, M.D., San Diego, Calif. 612

A Bilateral Posterior Intranasal Pack
Sidney H. Gidoll, M.D., San Francisco 615

New Instrument—Roller Knives
Harold F. Schuknecht, M.D., Detroit 619

PROGRESS IN OTOLARYNGOLOGY

Bronchoesophagology
F. Johnson Putney, M.D., Philadelphia 620

REGULAR DEPARTMENTS

Abstracts from Current Literature 647

News and Comment 658

Directory of Otolaryngological Societies 659

A. M. A.
ARCHIVES OF OTOLARYNGOLOGY

VOLUME 69 MAY 1959 NUMBER 5

COPYRIGHT, 1959, BY THE AMERICAN MEDICAL ASSOCIATION

EDITORIAL BOARD

GEORGE M. COATES, Chief Editor
1721 Pine Street, Philadelphia 3

RALPH A. FENTON, Portland, Ore.
ROBERT L. GOODALE, Boston
VICTOR GOODHILL, Los Angeles
CHEVALIER JACKSON, Philadelphia
JOHN D. KERNAN, New York
CARL H. McCASKEY, Indianapolis
JOSEPH SATALOFF, Philadelphia
ERNEST M. SEYDELL, Wichita, Kan.
GEORGE E. SHAMBAUGH JR., Chicago

J. F. HAMMOND, Editor, A. M. A. Scientific Publications
GILBERT S. COOPER, Managing Editor, Specialty Journals

SUBSCRIPTION RATES

Price per annum in advance, including postage: Domestic, $14.00. Canadian, $14.50. Foreign, $15.50. Price to students, interns, and residents, $8.00 in U. S. & possessions.

Single copies of this and previous calendar year, $1.50.

Back issues older than two years are available through Walter J. Johnson, Inc., 111 Fifth Avenue, New York 3. Future reprints of back issues will be available through Johnson Reprint Corporation, 111 Fifth Avenue, New York 3.

Checks, money orders, and drafts should be made payable to the American Medical Association, 535 North Dearborn Street, Chicago 10.

AMERICAN MEDICAL ASSOCIATION Publication

Published monthly by the AMERICAN MEDICAL ASSOCIATION. Editorial and Circulation Offices: 535 North Dearborn Street, Chicago 10, Illinois. Publication Office: Thompson Lane, Box 539, Nashville 1, Tennessee.

Second-class postage paid in Nashville, Tennessee.

CHANGE OF ADDRESS: When there is a change of address, the Circulation Office of the American Medical Association should be notified at least six weeks before the change is made. The address label clipped from the subscriber's latest copy of the publication and a statement of old and new address should be included. If there is a postal zone number, it too should be included in the new address. The instructions should state whether the change is permanent or temporary.

New from Sonotone...Pure-Tone Audiometers with a full-year warranty!

Light weight, easy to carry. Tough plastic case won't crack, dent or chip under rigorous portable use.

- **Outstanding performance**
- **Low cost**
- **Simple, speedy operation**

Sonotone's new Model 91 series of audiometers meets or exceeds all A.S.A. technical specifications of design and performance.

So confident is Sonotone of their quality that you get a warranty of *one full year*—not just 90 days. The price? Just compare for yourself. (You'll be delighted!)

And they're simple to operate—the most-used controls are handiest. There are Model 91 types for every service—perfect for doctors' offices, health departments, schools, industry.

FREE BROCHURE! Write for illustrated brochure giving many more details on Sonotone's new pure-tone audiometers.

Sonotone Corp.,
Department AZZ-59, Elmsford, New York

NAME_____

ADDRESS_____

CITY_____ ZONE____ STATE_____

Sonotone® CORP.
Department AZZ-59
ELMSFORD, NEW YORK

A leading manufacturer and distributor of transistor hearing aids

Instructions to Contributors

Articles, book reviews, and other materials for publication should be addressed to the Chief Editor. Articles are accepted for publication on condition that they are contributed solely to this journal.

An original typescript of an article, with one carbon copy, should be provided; it must be double or triple spaced on one side of a standard size page, with at least a 1-inch margin at each edge. Another carbon copy should be retained by the author.

The main title of an article may not contain more than eighty characters and spaces; a subtitle may be of any length.

The author's name should be accompanied by the highest earned academic or medical degree which he holds. If academic connections are given for one author of an article, such connections must be given for all other authors of the article who have such connections.

If it is necessary to publish a recognizable photograph of a person, the author should notify the publisher that permission to publish has been obtained from the subject himself if an adult, or from the parents or guardian if a child. An illustration that has been published in another publication should be accompanied by a statement that permission for reproduction has been obtained from the author and the original publisher.

Oversized original illustrations should be photographed and a print on glossy paper submitted. Prints of a bluish tinge should be avoided. Large photomicrograph prints will be reduced in scale unless portions to be cropped are indicated by the author. The author should submit duplicate prints of roentgenograms and photomicrographs with the essential parts that are to be emphasized circled, as a guide to the photoengraver.

Charts and drawings should be in black ink on hard, white paper. Lettering should be large enough, uniform, and sharp enough to permit necessary reduction. Glossy prints of x-rays are requested. Paper clips should not be used on prints, since their mark shows in reproduction, as does writing on the back of prints with hard lead pencil or stiff pen. Labels should be prepared and pasted to the back of each illustration showing its number, the author's name, and an abbreviated title of the article, and plainly indicating the top. Charts and illustrations must have descriptive legends, grouped on a separate sheet. Tables must have captions. ILLUSTRATIONS SHOULD BE UNMOUNTED.

References to the literature should be limited to those used by the autnor in preparation of the article. They should be typed on a special page at the end of the manuscript. The citation should include, in the order given, name of author, title of article (with subtitle), name of periodical, with volume, page, month—day of month if weekly or biweekly—and year. References to books must contain, in the order given, name of author, title of book, city of publication, name of publisher, and year of publication.

AMERICAN MEDICAL ASSOCIATION

535 North Dearborn Street **Chicago 10**

for anesthesia in quietness and confidence

VINETHENE®

VINYL ETHER FOR ANESTHESIA, U.S.P.

induction prior to ether—VINETHENE "... is more rapid and smooth than nitrous oxide and the excitement period is practically eliminated."[1] May be administered with closed technic.

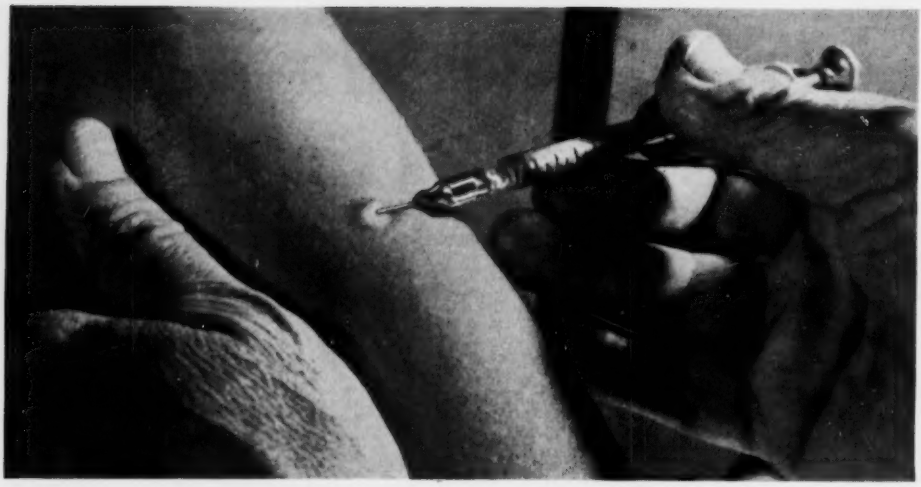
short operative procedures—VINETHENE is easily administered by open-drop method. Induction and recovery are pleasant and rapid, muscular relaxation is good, and excitement or nausea is rarely observed.

supplement to nitrous oxide or ethylene—VINETHENE complements ethylene-oxygen or nitrous oxide-oxygen mixtures and permits utilization of more oxygen. Excellent for brief surgical procedures.

1. Goodman, L. S., and Gilman, A.: The Pharmacological Basis of Therapeutics, ed. 2, New York, The Macmillan Company, 1955, p. 69.

supplied: In 10-cc., 25-cc., 50-cc., and 75-cc. bottles, each with adjustable plastic dropper cap.

MERCK SHARP & DOHME DIVISION OF MERCK & CO., Inc., PHILADELPHIA 1, PA. VINETHENE is a trademark of Merck & Co., INC.



when allergy tests are positive for

· food · dust · detergents · pollen · drugs

'Teldrin' provides "round-the-clock" relief from these "round-the-year" allergies.

Just one 'Teldrin' *Spansule* capsule q12h provides rapid, effective and prolonged relief from allergic symptoms. Side effects from 'Teldrin' therapy are minimal.

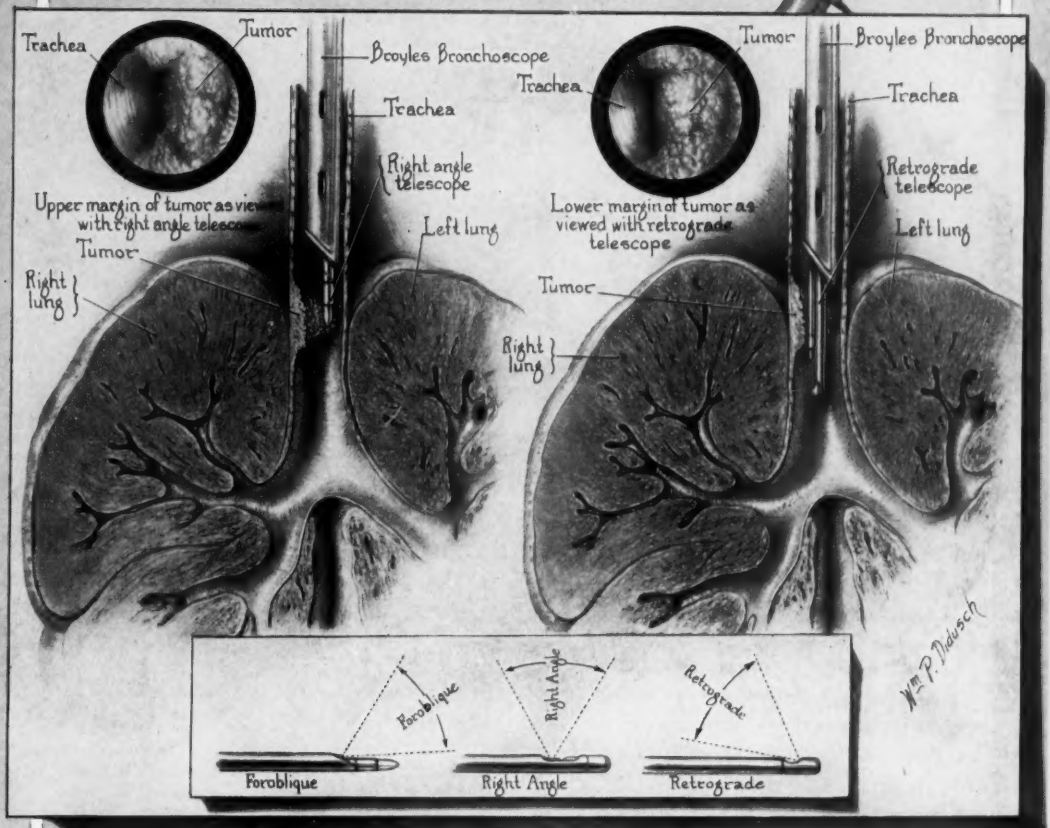
Available in 2 dosage strengths: 12 mg. for adults and older children; 8 mg. for younger children.

TELDRIN® brand of chlorprophenpyridamine

SPANSULE® brand of sustained release capsules

SKF SMITH KLINE & FRENCH LABORATORIES

THE *Broyles* BRONCHOSCOPE



The illustration above shows the Broyles Bronchoscope and the use of right angle and retrograde telescopes in evaluating a tumor of the trachea.

The Broyles Bronchoscope with the right angle, the foroblique, and the retrograde telescopes provides accuracy in the proper evaluation and subsequent treatment of lesions of the trachea.

Views heretofore never obtained are now possible with the combination of telescopes. Tumors can be outlined and the margins clearly defined, showing the degree of lateral spread and the characteristics of infiltration so vital in establishing a comprehensive study.

The telescopes accompanying the Broyles Bronchoscope are examples of fine optics and provide large clear images with a minimum of distortion. They are readily interchangeable and their use does not appreciably increase the period of examination.

ESTABLISHED IN 1908 BY REINHOLD WAPPLER

FREDERICK J. WALLACE, *President*

American Cystoscope Makers, Inc.

8 PELHAM PARKWAY PELHAM MANOR, N. Y.

9 SPECIALTY JOURNALS

PUBLISHED MONTHLY

BY THE AMERICAN MEDICAL ASSOCIATION

NEUROLOGY AND PSYCHIATRY

DISEASES OF CHILDREN

INTERNAL MEDICINE

INDUSTRIAL HEALTH

OTOLARYNGOLOGY

OPHTHALMOLOGY

DERMATOLOGY

PATHOLOGY

SURGERY

each journal offers
the latest medical findings by
outstanding authorities in
its special field . . .
of value not only
to the specialist but
to the general practitioner as well

to order your subscription to one of the A.M.A.'s
specialty journals use the form below

AMERICAN MEDICAL ASSOCIATION
535 North Dearborn • Chicago 10

Please enter my subscription to the specialty journal checked at right.
Start my subscription with the next Issue.
Remittance for ☐ one year ☐ two years is enclosed.

NAME_____

ADDRESS_____

CITY_____ ZONE____ STATE_____

	U.S.A. & Possessions APO's	Canada	Outside U.S.A. & Possessions
☐ A.M.A. Arch. Neurology and Psychiatry	$14.00	$14.50	$15.50
☐ A.M.A. Arch. Dermatology	12.00	12.50	13.50
☐ A.M.A. Arch. Industrial Health	10.00	10.50	11.50
☐ A.M.A. Arch. Internal Medicine	10.00	10.50	11.50
☐ A.M.A. Jrl. Diseases of Children	12.00	12.50	13.50
☐ A.M.A. Arch. Surgery	14.00	14.50	15.50
☐ A.M.A. Arch. Pathology	10.00	10.50	11.50
☐ A.M.A. Arch. Ophthalmology	12.00	12.50	13.50
☐ A.M.A. Arch. Otolaryngology	14.00	14.50	15.50

new SUPER HEADLIGHT

BY GOOD-LITE

- COOLER OPERATION
- 9 INTENSITIES OF LIGHT
- INCREASED SAFETY— UNDERWRITERS APPROVED

Medical men who have tried the new Super Good-Lite tell us there's nothing like it anywhere . . . at any price! Here's why: The entire unit has been redesigned by a group of *practicing surgeons* to provide a beam of light both brilliant and controllable . . . featuring nine intensities of light. The headlight construction and electrical circuit was designed for cooler operation and maximum safety.

The Good-Lite SUPER Headlight Includes:

- Good-Lite parallel beam headlight.
- 3 types of bulbs with 3 intensities of light each.
- Special Ground lens to intensify light.
- 2 mirrors (peep-hole and plain).
- Heavy duty transformer (20 watt) with 3 positions and off switch. Fuse in the secondary for your safety.
- Molded plastic connections.
- Gray plastic stainproof cords.
- Choice of 5 types of headbands.

Consult Your Surgical Supply Dealer or Send Coupon Today

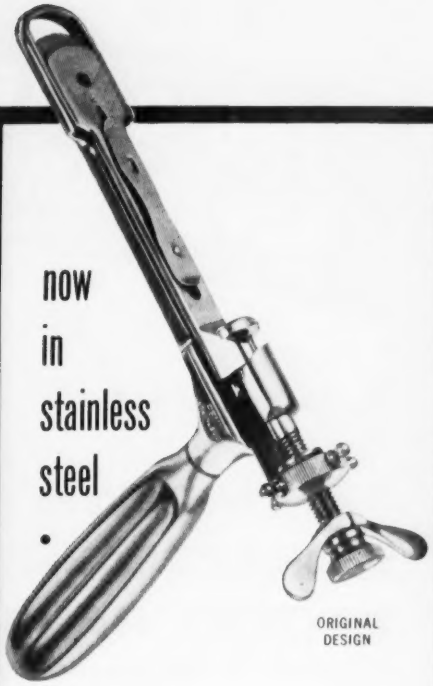
GOOD-LITE MFG. CO.
7638 W. Madison St. • Forest Park, Ill.

Please send free catalog on Good--Lite Headlights to:

Dr. _____

Address _____

City _____ Zone _____ State _____



now in stainless steel

ORIGINAL DESIGN

DANIELS'

HEMOSTATIC TONSILLECTOME

This proven instrument for the bloodless enucleation of tonsils is now available in stainless steel. Improved metallurgy combined with mechanical perfection assures long life.

Available in three sizes. Ask your surgical supply dealer for illustrated literature or write. All Dittmar-Penn Instruments are sold only through recognized surgical supply dealers.

Dittmar·penn

809 N. 19th St., Phila. 30, Pa.
MANUFACTURERS AND WHOLESALERS

END BATTERY REPLACEMENTS

New!

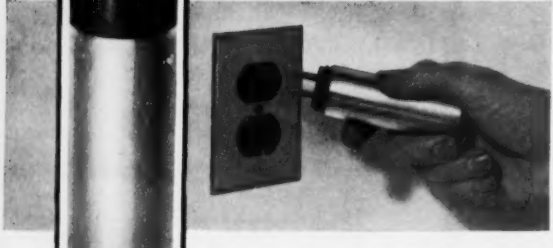
WELCH ALLYN RECHARGEABLE BATTERY HANDLES

with desk or wall mounted charger

- **Always fully charged in office use.** Place handles in charger when not in use and they recharge automatically. Can't overcharge.
- **Last as long as large conventional batteries,** without recharging, when used on house and hospital calls. Can be recharged thousands of times.
- **Handles are small and lightweight,** even smaller than WA medium (No. 705) handle.
- **Compact, attractive charger** takes only 7½" x 4" space on desk, or installs on wall bracket. Plugs into 110 v. AC outlet.
- **Handles have Permafit collar,** accept any WA instrument head.

WA No. 712 Two rechargeable handles with desk-type charger (less instrument heads) $60.00

RECHARGEABLE BATTERY INSERT FOR WELCH ALLYN LARGE SIZE HANDLES



This new combination battery and charger slips into your present large (WA No. 700) handle. No separate charger required. Lasts as long as conventional batteries without recharging.

Recharges automatically when plugged into 110 v. AC outlet. Can't overcharge. Can be recharged thousands of times.

No. 719 Rechargeable battery insert for No. 700 handle $15.00

WELCH ALLYN

LIGHTS THE WAY

A.M.A. ARCHIVES OF
OTOLARYNGOLOGY

Principles Used in the Development of Modern Temporal Bone Surgery

JULIUS LEMPERT, M.D., New York

History

Modern otology can be said to have its origin in the 18th century. It is then that the first attempts were made to articulate principles which might guide the ear surgeon. In the light of later learning we see that in some instances the precepts of that day were not correctly formulated, nor were they clearly understood or applied. Surgical practice of that time seems to have swung between principles we now know are incorrect and those to which we still adhere. Even those views which later proved erroneous, however, had their place in the development of aural surgery and will be discussed briefly .

The 18th Century.—Jean Louis Petit, working in France in the 1750's, had operated successfully for suppurative mastoiditis by trephining the mastoid process. It was confirmed in his published papers (which, however, came out after his death) that he understood this important principle, namely, that purulent inflammation of the mastoid is the only indication for surgical intervention.

If this important point was known to other otologists of that day, it was overlooked and not applied by the many surgeons who, some 20 years later, adopted the so-called "Jasser operation." Jasser, a Prussian military surgeon, working in the 1770's, found by accident that rupture of the drum in an infected ear and spontaneous escape of fluid improved a patient's hearing. He thereafter advocated an opening in the mastoid for improvement of hearing. Evidently he and some of his contemporaries believed that deafness could be alleviated by establishing a pathway to the middle ear through a perforation in the mastoid process (misinterpreting the importance of drainage of pus) to permit sound to enter the middle ear through the mastoid antrum. The "Jasser operation" became widely used as a means for curing deafness. Its misapplication caused many unfortunate results. The most notable, of course, was the case of Baron von Berger, Danish court physician, upon whom the "Jasser operation" was performed in 1791 and who died of meningitis some days after this surgery.

Following the reports of the Berger incident, aural surgery received a setback which took many years to overcome. By the end of the 18th century it had been temporarily forgotten, not to be revived until more than a half century later.

The 19th Century.—The early years of the 19th century produced many distinguished otologists. Otology was becoming recognized as a separate branch of medicine.

Submitted for publication Dec. 12, 1958.

Lempert Institute of Otology, Lempert Research Foundation, Inc.

Wherry Memorial Lecture, presented before the 63d Annual Session of the American Academy of Ophthalmology and Otolaryngology, Chicago, Oct. 12-17, 1958.

Its more important developments were not in surgery of the ear, although contributions to knowledge of the anatomy and physiology of the ear provided a firm background for further advances in surgery. (Cotugno's description of the complexity of the inner ear had already come into the literature and it was known, for example, that the labyrinth is filled with fluid.) Virchow became world-famous for his studies of pathology; Helmholtz formulated his theories of sound transmission which were to become so vital to the field of otology. Advances in bacteriology by Pasteur, in antisepsis by Lister, and the introduction of anesthesia, all played an important role in the establishment of aural surgery on firm bases.

In England, Joseph Toynbee, one of the great pioneers in scientific otology, was engaged in his dissection of temporal bones, and recorded his findings in his book, "Diseases of the Ear," published in 1860. To insure that his descriptions would be as exact as possible he dissected more than 2,000 temporal bones. He showed how to remove the temporal bone, and compared morbid appearances with symptoms noted during life. Meanwhile, in Ireland, William Wilde, the first true "ear specialist," described the mastoid incision which came to be known as the "Wilde incision." It was widely used and made him famous. Although it was based on erroneous anatomical and surgical knowledge, still its long popularity seems to have helped in keeping alive the practice of mastoid surgery. It was long after Wilde's day, of course, that this important maxim became accepted: In any case in which the "Wilde incision" might be indicated, a mastoid operation is absolutely essential.

Among the more outstanding advances made in the 19th century were von Troeltsch's contributions, in Germany. In a detailed paper, published in 1861, he gave a precise description of his mastoid operation and its indication. He wrote, "One of the fundamental principles of surgery, which is slowly gaining ground in internal medicine as well, demands that every collection of pus in the tissue should be evacuated externally as soon and as completely as possible. In this way only can damage to neighbouring structures, gravitation abscesses, and extension of inflammation be avoided." It is difficult to understand why prior to von Troeltsch's time aural suppuration was not considered an indication for surgical intervention. Otologists before that relied on perforation of the mastoid cortex and merely hoped for drainage, whereas surgical intervention for pus accumulation in other parts of the body was becoming an accepted procedure.

Von Troeltsch's article gained wide attention and in the next few years a number of other surgeons contributed papers to the literature on mastoid surgery, indicating complete acceptance of von Troeltsch's views. In 1868, Jacoby, an internist in Breslau, commented that "before long the operation of perforation of the mastoid will be adopted as the treatment of specially obstinate middle-ear suppurations, even when no urgent symptom of retention of pus is present." In 1873 Schwartze's reports of his early cases of trephining the mastoid process were published. At first he had used a perforator but then discarded this in favor of chisel and hammer.

Starting in the late 80's there were further significant advances in mastoid surgery, on which the present day mastoid operation is still based. Augmenting Schwartze's work, Küster advocated "opening up the bone freely so that it can readily be scrutinized to clear away all disease and fully expose the source of suppuration." Zaufal, in 1884, wrote that he had been led by Küster to adopt removal of the posterior wall of the meatus and he gave a detailed description of seven steps in his procedure. In 1897 Stacke's book, "On the Operative Exposure of the Middle Ear Cavities," appeared. In Stacke's operation the attic is opened by chiseling away the pars epitympanica and passing a probe into the antrum, thus opening the cavity from the meatus and the external wall. He removed the malleus. By

the end of the 19th century the principles of modern mastoid surgery, based on Schwartze's operation, as modified by Stacke, became the model on which mastoid surgery for otherwise incurable chronic middle-ear suppuration, was based.

No résumé of temporal bone surgery in the 19th century would be complete without the name of Adam Politzer. Working in Vienna, he was becoming internationally famous for his contributions to the field of otology. Among his chief original additions to treatment of ear disease was his method of inflation of the middle ear ("politzerization") which is now largely abandoned; but his description of the intracranial and other complications of otitic diseases was of lasting assistance to ear surgeons.

Politzer's outstanding publications included the famed Otoscopic Atlas and his book on diseases of the ear, which became a classic and is still counted one of the most important works on the ear ever to be printed. His monumental two-volume "History of Otology," almost encyclopedic in scope, is an invaluable part of the literature on the ear. It is perhaps less well known in the United States than it deserves to be, since it has not been translated into English. The volumes cover comprehensively the entire history of the anatomy and physiology of the ear and of physics of sound and hearing, up to the year 1911.

The most significant of Politzer's contributions, however, was his role as a teacher. As the first professor of otology at the University of Vienna, from 1870, he drew to his clinic physicians from all over the world. It has been said that over 7,000 postgraduate students attended his classes, which he taught equally well in German, Italian, French, and English. The roster of names of his students reads like a "Who's Who in Otology." It includes among other respected otologists: Neumann, Ruttin, Leidler, Alt, Bárány, Gradenigo, Lucae, Lermoyez, Alexander, Panse, Denker, Hartmann, Bezold, and dozens of other Europeans. Politzer can be credited with having lasting influence on the development of otology in the United States also, for it is certain that Clarence Blake, of Boston, one of the great American otologists, was indebted to Politzer, whose assistant he was for a time in Vienna. Another outstanding American otologist, Gorham Bacon, of New York, was also a Politzer pupil.

The last decade or so of the 19th century saw the introduction of another phase of temporal bone surgery. This was attempted mobilization of the stapes, a surgical process that occupied otologists in Europe and the United States in the closing years of the 19th century.

In order to indicate the rationale for attempted mobilization of the stapes as a means for restoring hearing, prior to the 1890's, it is necessary to look back for a moment to the 18th century. Ankylosis of the stapes had been mentioned by Valsalva as early as 1735. Morgagni, in 1766, also mentioned a case in which the fenestra ovalis and the base of the stapes formed a solid bony plate; Meckel, in 1777, described one in which the membrane connecting the base of the stapes with the fenestra ovalis had become bony. Toynbee, in 1841, was among the first to make a distinction between bony and membranous ankylosis on the basis of pathological findings in his dissections of temporal bones. Schwartze, in 1870, also demonstrated in a number of autopsied temporal bones from persons known to be deaf during lifetime, that stapes ankylosis was the cause of the hearing loss. Schwartze credited von Troeltsch with the term "sclerosis" resulting in ankylosis of the stapes. Bezold (1885), Gradenigo (1887), and Politzer (1882) all described stapes ankylosis, resulting either from adhesion of the footplate to the margin of the oval window, or by attachment of the crura to the lower wall of the niche of the oval window. In 1891 Haberman reported pathological findings in a postmortem case, and in 1897 Panse recapitulated the findings of the foregoing investigators. He had studied the temporal bone collections of Toynbee, Politzer, and Haberman, and pub-

lished a monograph in which he maintained that the stapes and the round window are the most important parts of the sound-conduction chain; that rigidity of these structures is usually the cause of hearing loss.

Accepting stapes ankylosis as a proved cause of deafness, Kessel, as early as 1876, had recommended that the tympanic membrane, the incus, and malleus be removed if the ankylosis was the result of suppurative processes. It was Kessel who also suggested that if attempts to mobilize the incudostapedial joint or footplate of the stapes are successful without disrupting the ossicular chain, the improvement of hearing in cases of "sclerosis" may be expected.

In 1888 Boucheron reported on 60 cases operated on for mobilization of the stapes, the operation consisting of separation of the incus from the stapes, excision of the posterior half of the tympanic membrane, mobilization of the stapes by gentle traction by means of a hook, moving the stapes back and forth and up and down by traction on the stapedial muscle, and in some cases, removal of the incus and the malleus.

In 1890 Miot, in a report based on 126 cases with successful results in 74, concluded that in deafness without severe thickening of the tympanic membrane, in the absence of paracusis but with diminution of cranial perception and tinnitus, mobilization of the stapes gives good results even when the condition has persisted for several years.

In 1892 and 1894 Jack reported on 66 cases of stapedectomy, with "discouraging" results, concluding that the operation is not advisable in cases of sclerosis and that "most operations for mobilizing the stapes must be looked upon as largely experimental." In 1893, Politzer, and in 1894, Kessel reported that neither tenotomy, mobilization, nor extraction of the stapes can be expected to improve hearing; Panse, Vulpius, Clark, Kaufman, Alt, and others, in the years from 1896 to 1898 reported indifferent results with their attempts to mobilize the stapes or with stapedectomy. A new facet was introduced in the year 1898, when Alderton, of Brooklyn, having tried mobilization without success, and later a stapedectomy in the same case, finally resorted to trephining the footplate. Labyrinthine fluid was seen to escape, according to his report, and there was no dizziness, but three weeks later the fenestra ovalis was hidden by cicatricial tissue. Tinnitus and hearing were the same as before the operation.

In 1897 Politzer summarized current thinking on stapes mobilization thus:

"The many attempts by Kessel, Boucheron and Miot to mobilize and remobilize the rigid stapes in the niche of the oval window, which sometimes had a temporary but short lived improvement of hearing, is completely abandoned.

"The same is true with the extraction of the stapes which was tried out by Kessel, Jack, Dench and others. Experiments on animals in this direction were made by Botey, Faraci, Politzer and others. Blake, Knapp, Cheatle, and Politzer do not advocate this operation."

By 1900 Siebenmann reported that all attempts at mobilization of the stapes in "sclerosis" are not only useless but often harmful, and that "avulsion of the stapes is no more successful." (The term "otosclerosis" seems to have been introduced about 1893, but in Siebenmann's works had not yet come into use.)

The 20th Century.—There the matter seemed to rest. No reports of further surgery for stapes fixation came from the great otologists of the early years of the 20th century. Politzer, Neumann, Leidler, Ruttin, Blake, Jack, Dench, all remained silent. There are almost no reports on record of further attempts to remobilize the ankylosed stapes until over 50 years later.

But this is not to suggest that progress was at a standstill in otology. Many important advances were made in the development of the radical mastoidectomy, physiology, and pathology and in the understanding of the physics of sound.

Attempted surgery for improvement of hearing being temporarily forgotten, mastoid surgery again became the primary concern of otologists. Emil Gruening was the first in the United States to publish a thoroughly detailed and precise analysis of mastoid surgery, basing his procedure on Schwartze's original technique as modified by Stacke, but adding further modifications of his own. Gruening seems to have been the first to advise that when the mastoid process harbors purulent inflammation extending beyond the antrum and involving the body of the bone, the tip should be removed as a routine matter. In 1905 Whiting advocated still another modification of the Schwartze-Gruening procedure, i. e., that all cellular structure from within the mastoid process, including the cells within the mastoid tip, must be exenterated during the performance of a simple mastoidectomy.

About this time too, early in the 20th century, Edward B. Dench's precise indications for the radical mastoidectomy helped to establish a basic procedure in aural surgery. Emanuel Libman, world-famous American internist, in 1911 contributed to ear surgery by pointing out that in jugular sinus thrombosis, a positive blood culture is obtainable. The following year, Alfred Braun added to knowledge useful to ear surgeons by publishing a comprehensive treatise on the pathology, symptomatology, and surgical treatment of sinus thrombosis.

In physiology and pathology the early 1900's saw the addition of these important observations: Following Bárány's notable publication in 1907 on the physiology of the vestibular labyrinth, Isador Friesner and Alfred Braun, in their book published in 1916, presented a basic and accurate picture of clinical labyrinthitis. Early in the 1920's, George E. Shambaugh Sr., through his painstaking histologic studies gave new understanding to the anatomy and pathology of the inner ear. This highly significant work was augmented by Stacy Guild's investigations in correlation of the histological findings with the clinical findings in inner-ear diseases. Also in this period Sir Charles Ballance, forming a team with Arthur B. Duel and working in this country, conducted significant animal experiments in nerve grafting. Their researches led to presentation of the famous Ballance-Duel operation for facial nerve injury.

However, as important as the Ballance-Duel contribution was to otology, it must be admitted that a much better aid to otologic surgery and to humanity would have been a means for providing better training in temporal bone surgery, so that facial nerve injury would be less frequent. If, in spite of better knowledge, facial nerve injury accidentally occurred, the surgeon would know when, where, and how it happened. With such understanding, an occasional injury to the facial nerve would be easily overcome. As a rule it could be corrected immediately, on the operating table, and permanent facial paralysis requiring the Ballance-Duel operation would be avoided.

In the same decade, Wells P. Eagleton's extensive investigations for unlocking the petrous pyramid of the temporal bone, in suspected presence of infection within the petrous apex, pointed the way to prevention of many cases of suppurative meningitis following temporal bone operations. In this same period, Samuel J. Kopetzky systematized the description of the symptoms of suppurative petrositis, and Ralph Almour described a technique for perforating and draining the infected petrous pyramid.

In 1928, publication of my paper, "Simple Subcortical Mastoidectomy," laid the foundation for the gradual development of the techniques for the endaural approach to all temporal bone surgery, and became an available surgical procedure for drainage of the infected petrous pyramid.

This paper, the first, I believe, to describe the subcortical simple mastoidectomy technique, engendered immediate and widespread interest throughout the profession. In the years following the establishment of my own hospital in 1923, I was developing the endaural technique and had shown its

advantages as the most direct approach to the temporal bone. It was also proving to be the most reliable approach to the mastoid antrum, without searching for it, and without danger of injury to any of the vital anatomy. This paper also served to dispel the erroneous view that had gained some currency in the literature that in some cases there is an absence of the mastoid antrum. In the many thousands of mastoid operations which I performed before then, and have done since the publication of that paper, I have never encountered a temporal bone in which the mastoid antrum was absent! This paper also recommended use of the drill to supplant the hammer and chisel.

It was to be expected that rebuttal arguments would be made. A number of articles appeared in the literature claiming that complete mastoid-cell exenteration was impossible with the endaural approach. In 1928, on invitation of Professor Le Mee, I went to Paris to demonstrate this new procedure, and did so, at the Blumenthal Pavillon of the Hospital des Infantes Malades. Next, at the invitation of Professor Voss, in Frankfurt-am-Main, I again demonstrated the application of the endaural technique and then, at the invitation of Dr. Klaus, presented my paper on this technique at a meeting in Berlin. This was followed by a visit to Vienna, where, at the invitation of Neumann, Politzer's successor at the University of Vienna, I operated at the Wiener Allgemeines Krankenhaus. Before a group of distinguished European otologists I again performed a mastoid operation to demonstrate the reliability of the endaural approach and went on to show that all related complications could be handled successfully through the endaural route—sinus thrombosis, middle and posterior fossa abscess, and suppurative labyrinthitis. One of the patients entrusted to me by Neumann was a young child with a Bezold abscess. I demonstrated to the group that such infections could be taken care of successfully through the endaural route.

The following year, 1929, at the invitation of Professor Le Maitre, I read a second paper on "Subcortical Mastoidectomy," and showed films, at the French Congress of Otolaryngology. Also at Le Maitre's invitation, I demonstrated my procedure at the Hospital St. Louis. Then came an invitation from Bruhl, and I showed the procedure at the Maria Victoria Krankenhaus. Again at the Wiener Allgemeines Krankenhaus at Neumann's invitation I showed this surgery in Vienna. At a meeting of the Wiener Gesellschaft der Arzte which followed, Neumann did not hesitate to report that he was convinced of the efficacy of the endaural procedure for all mastoid surgery. "Everything can be seen," he said, "and where you see everything you can do everything." Thereafter, Beyer, of the Charité Hospital in Berlin, reported his acceptance of the endaural approach and was the first to include a description of it in his textbook on mastoid diseases and their surgical treatment. Leidler, after the Vienna meeting, reported favorable results on a series of cases for which he used the endaural route.

My demonstrations in Europe proved the usefulness of the endaural approach for exenteration of infection within the petrous pyramid. A number of other papers appeared, among them those of Voss, in 1932, and Ramadier, in 1933. Each described a method for perforating and draining infection in the petrous apex. In 1936, publication of my paper, "Complete Apicectomy," brought into the literature complete directions for deliberate removal of all the cell structure with its pathology from within the diseased apical portion of the petrous pyramid.

The next important milestone in the history of mastoid surgery was reached, I believe, with my paper, "Endaural, Antauricular Surgical Approach to the Temporal Bone," published in 1938.

These four papers (published in 1928, 1929, 1936, and 1938) furnish what in my opinion is a reliable guide for the use of the endaural approach whenever surgical

intervention in the temporal bone is necessary—for all mastoid diseases, including suppuration within the petrous pyramid. The basic surgical principles for the endaural approach permit the following:

1. Complete exenteration of the pathologic processes with a minimum of trauma, a minimum of healthy tissue sacrifice, and with the least permanent postoperative deformity.
2. Step-by-step methodical exenteration of the cellular temporal bone structure, for deliberately planned, instead of accidentally encountered, visualization of all the vital temporal bone anatomy.
3. With proper lighting and magnification, direct and methodical exposure of all the vital anatomy, give added assurances against the possibility of overlooking any pathology within the temporal bone or of accidentally injuring any of the vital anatomy.

With these principles in mind, I believe that any well-trained aural surgeon has a complete index of safe and comparatively simple mastoid techniques.

Development of the Fenestration Operation

The viable tympanomeatal pedicle flap, which I employed for sealing a labyrinthine fistula in the performance of the endaural mastoidoatticotomy for chronic epitympanic suppuration, led ultimately to my development of the practical one-stage fenestration operation for restoration of hearing in clinical otosclerosis.

Since all the important contributions to fenestration surgery were made in the last three decades, perhaps the historical background is known to most of you. However, a brief review of the chronology may add a point or two to the knowledge of this important work.

It is customary when describing the origin of operations for otosclerosis to start with the names of Kessel, Politzer, Passouw, and Panzer, concluding with a description of the work of Katz, who is given credit for the first full report of the pathology of stapes ankylosis, about 1890. There was obviously a tie between the recognized symptoms of the disease and the attempts to overcome them in the methods of treatment employed by these earlier aural surgeons. For example, Politzer devised his method of inflation for the express purpose of attempting to mobilize the ossicular chain.

The first deliberate attempt to make a new vestibular window, surgically, apparently came from Jenkins, who in 1913 opened the semicircular canal in the belief that otosclerosis was due to changes in density of the labyrinthine fluid. Evidently he believed further, that opening of the canal would alter the condition of the fluid. In his operation no fluid was seen to escape; the hearing improved only slightly at first, then retrogressed. Fraser, in Edinburgh, in 1919, is also reported to have experimented with opening the horizontal canal. Next, Bárány operated on several patients for otosclerosis by opening the posterior bony canal. Although immediate improvement of hearing was noticed, it did not last. In 1923 Holmgren reported on his attempts at surgical treatment of otosclerosis, his innovation being the introduction of the binocular microscope. In three of four cases reported, hearing was improved.

The many failures and accidents that followed fenestration attempts in England apparently made it necessary to defer further trials of this surgery there. But the pioneers never entirely gave up. It had been proved beyond doubt that an opening in the semicircular canal would restore hearing if the deafness were caused by otosclerosis. Even a pinpoint opening would serve the purpose. The only problem which remained to be solved was how to prevent osteogenesis, with subsequent closure of the newly made opening.

To Gunnar Holmgren's persistence on the Continent, in the face of failure, may be attributed the eventual success of the fenestration operation and the fact that we have it today as a means for restoring hearing in clinical otosclerosis. When Maurice Sourdille set to work on the problem in the 1920's, it was through Holmgren's inspiration as a teacher, and the inspiration flowed both ways. Sourdille obviously inspired Holmgren to continue his efforts.

It was in 1924, after a visit to Holmgren, that Sourdille described his two-stage operation which he called "tympano-labyrinthopexy." In this procedure he performed a mastoidoatticotomy, retained the incus, removed the head of the malleus, and skin-grafted the mastoid wound. After the mastoid wound had completely healed, he elevated the thin scar, and opened the horizontal canal. The scar, which became attached to the tympanic membrane, was replaced over the new window. An interval of six to eight months was necessary between the two stages of the operation. In a report of the New York Academy of Medicine in October, 1937, he gave the results in 109 patients, reporting 40% success, based on hearing for the whispered voice. He said that his main cause for failure was osteogenesis, with closure of the window, and in some cases, injury to the membranous canal.

In a letter dated April 12, 1938, read at a meeting of the American Otological Society, Holmgren said: "It may be possible to obtain permanently open fistulas of the labyrinth, but until now this has been the case only occasionally, with Sourdille's material and with mine, and there is no known method for reaching this goal."

In 1938, with the publication of my paper, "Improvement of Hearing in Cases of Otosclerosis," there began a continuity of effort to perfect a technique for a one-stage fenestration operation that would provide for permanent improvement of hearing in clinical otosclerosis. This, I feel, has been achieved. With the Lempert fenestration operation, using what I term the nov-ovalis dry technique, I believe we have the means of giving practical, serviceable hearing to 90% of the suitable cases of clinical otosclerosis. Suitability for the procedure can be determined preoperatively. I have now performed over 10,000 fenestration operations. Some of my patients have been watched for as long as 20 years, and still have hearing that has been improved to the level where they can hear ordinary conversation without a hearing device; they have been restored to normal, happy lives, and are useful members of the community.

The foremost principles on which I based my surgical procedure for the fenestration operation are these:

Principle 1. Any technique employed to improve hearing in clinical otosclerosis must provide for the possibility of remobilizing the inner ear fluids by air-borne sound sufficiently to permit the restoration of practical, serviceable hearing, without the need of a hearing aid. The surgical research which I have carried out since the inception of the fenestration technique in 1938 has convinced me that to obtain such a degree of remobilization of the inner ear fluids a new covered fenestra must be created outside of the tympanic cavity, namely, in the external semicircular canal, to replace the covered intratympanic oval window functionally impeded by the otosclerotic growth. In 1950, Wever and Lawrence, reporting in the *Transactions of the American Otological Society* on the results of investigations of sound conduction in animals, confirmed the correctness of my theory that improvement of hearing following the fenestration operation depends upon the successful remobilization of the inner-ear fluids. They said, "The fenestration operation, as we have seen, effects a remobilization of the cochlear fluid." As long as means for arresting the growth and spread of the otosclerotic tumor within the tympanic cavity remain unknown, the permanent maintenance of hearing improvement obtained by any surgery within the tympanic cavity will not be possible. To prevent the possibility that the continuously growing, and uncontrollable, otosclerotic tumor may become a threat to continuity of improved hearing, placement of the new fenestra outside of the tympanic cavity is an absolute "must."

Principle 2. In the use of any technique for the improvement of hearing by reconstruction of the air-conduction mechanism, the biological, physiological and physical factors involved in providing the continuous

maintenance of such surgical reconstruction must be heeded.

Other basic features of the fenestration procedure are the following:

1. It includes means for predicting preoperatively the possibility for improving the hearing for air-borne sound to at least the 30 db. level in the speech frequencies—512, 1,024 and 2,048. The ability to hear at these levels is necessary for serviceable, unaided hearing. Normal hearing, following any surgical technique used for the improvement of hearing in otosclerosis, is rarely attained. Reports of such improvements will remain questionable until such time as audiometers are standardized by the Bureau of Standards.

2. The surgical technique employed is capable of improving hearing in any patient, no matter how low the preoperative audiometric reading indicates air-borne sound perception to be in these three frequencies, so long as the masked bone-conduction hearing for pure tones in these frequencies has not fallen below the 20 db. level.

3. The technique used incorporates a known, definitely prescribed, and time-proved means for deliberately conserving and permanently maintaining the hearing gain newly obtained by the operation. This is accomplished by using the invagination principle in covering the new fenestra, which prevents its osteogenetic closure and thus provides for its permanent functioning.

4. The procedure incorporates precautionary measures against any future labyrinthine infection secondary to middle-ear infection which may occur during the lifespan of the patient. This is accomplished by placing the new fenestra outside the middle ear, covering and sealing it off without leaving any direct connection between it and the middle ear.

5. The operative technique makes use of the physiological principle known to govern acoustical function of the inner ear following fenestration surgery. For this, also, placement of the new window outside the middle-ear, covering, sealing it off without leaving any direct connection between the new vestibular window and the middle ear, accomplishes the purpose. This serves to vary the sound pressure acting on the round cochlear window and on the newly created vestibular window. Thus, sound pressure reaching the intratympanic cochlear window is relatively less than the sound pressure acting on the newly created extratympanic vestibular window, and this provides the phase difference essential to mobilize the inner ear fluids by air-borne sound.

In connection with the surgical principles on which fenestration operation is based, some important contributions outside the field of surgical research should not be overlooked.

Among the investigations carried out by Ernest Glen Wever were those designed to show the results of such abnormal conditions as closure of either of the two windows. With the use of the precise methods he devised for determining the electric potentials of the ear and recording the actions of the ear in response to sound, he has been able to describe what happens when any one of the ossicles is disturbed or what occurs when the semicircular canal is opened in the performance of the fenestration operation. It was through these studies that he was able to show the correctness of my theory that remobilization of the inner-ear fluids is essential for obtaining the improvement of hearing. In his recent book, "Physiological Acoustics," written in collaboration with Merle Lawrence, Wever confirmed the physiological principles underlying the fenestration operation as now employed by me and as generally adopted by otologists throughout the world.

Other significant studies in the theory of hearing which have practical application in ear surgery were made by Georg von Békésy. With the development of his instruments which permit measurement of the vibrations of the eardrum and ossicles, and with further devices which follow the transmission of energy from the free sound field entering the ear, he is able to measure the reaction of sound on the innermost cells

of the end organs. His invention of the "Békésy audiometer," which provides for automatic testing of hearing, removes from audiometry the probability of human error, and makes for more scientific testing.

The Lempert fenestra nov-ovalis operation was developed by observation and use of all applicable physiological, biological, and physical principles.

Laboratory studies carried out by me with the assistance of Dorothy Wolff have made it possible to assemble the largest exhibit of histological and microscopic sections on the temporal bone anywhere in the country in relation to both otosclerosis and to Ménière's disease. My exhibit of sections of the ossicles proves the unmistakable presence of otosclerosis in the ossicles.

The Work on Surgical Procedures for Ménière's Disease

The 20th century contributions to temporal bone surgery as it relates to Ménière's disease begins with the observations reported by Hallpike and Cairns in 1938, that hydrops of the labyrinth is the cause of this disorder. For a time thereafter, "labyrinthine hydrops" became a term of reference for Ménière's symptom-complex, but whatever the terminology, surgical procedures designed to overcome the vertigo, symptomatic of the disease, were developed. An important surgical principle was set forth when Dandy described his operation, in 1941. Dandy's method involved exposing the eighth nerve through an opening in the posterior fossa; then the vestibular portion was separated, and only that portion was severed in the hope of saving the hearing. To my mind Dandy's procedure would be a valuable adjunct to temporal bone surgery, except that there exists an intense popular antipathy to brain surgery. The true diagnosis of Ménière's disease rests on the fact that each attack of vertigo causes additional loss of cochlear nerve function. Today it is the rule not to operate in Ménière's disease until such time as the hearing loss in the involved ear is so severe that it is no longer useful.

Admittedly the other forms of surgery introduced for Ménière's disease are no cure for it, since they involve destruction of the residual hearing and the balance mechanism in the affected ear. However, contributions to methods for relief of the vertigo and imbalance were made by Cawthorne, through removal of the membranous labyrinth, and by Day, who performed a simple mastoidectomy, exposing the horizontal canal, and made an opening into the labyrinth through which a mild coagulating current was introduced.

The contribution of Lempert to the surgical treatment of Ménière's disease consists of the operation for removal of the stapes and opening the round window membrane to permit the perilymph to escape gradually, so that there will be further and finally total degeneration of the membranous vestibular labyrinth. The technique used is the transtympanic approach which I first developed for use in middle-ear surgery, when searching for and removing pathology sometimes found in the hypotympanum in persistent cases of sero-otitis, and later used in the tympanosympathectomy procedure for relief of intractable tinnitus aurium. With this procedure it is possible to dislodge a part of the annulus fibrosus of the tympanic membrane from the sulcus tympanicus and to replace the membrane in its normal position without injury to it. It is this procedure which has now been adopted as the first step in the stapes mobilization operation by those who are practicing this surgery today.

A theory of the etiology of Ménière's disease has been advanced by me as a result of studies of the membranous vestibular labyrinth removed in vivo during the performance of partial labyrinthectomy for the arrest of vertigo in this disease. I believe these to be the first such investigations of human labyrinths undertaken immediately after this surgery, during the patient's lifetime. Histological studies conducted by Dorothy Wolff on the series of over 30 such labyrinths removed lead me to believe that the disease is of virus origin, related to

herpes. We have shown the similarity between the lesions found in these diseases. We believe that Ménière's symptom complex is the result of epithelial vesiculation, that the vertiginous attacks occur when one or more vesicles rupture with release of fluid toxic to the labyrinthine contents. Further research is being carried on at the Lempert Institute of Otology in this field. For assistance in this research the Lempert Institute has a grant from the United States Public Health Service.

Some Pros and Cons of Antibiotic Therapy

The introduction of the chemotherapeutic drugs and the antibiotics had advantages and disadvantages.

It is obvious that in the treatment of ear diseases antibiotic therapy was of enormous value, even becoming life-saving in meningitis and intracranial complications of otitic origin. It is also true that the safety provided by antibiotics made it possible to develop some of the current surgical procedures, for example, the one-stage fenestration operation.

One great disadvantage, however, seemed to be that of distorting the surgeon's judgment, especially with respect to otitis media. For a time it seemed that the usage of antibiotics had led to a permanent and unjustified abandonment of an important surgical principle—the provision for drainage of infection wherever found in the body. Complete reliance on antibiotics led to much difficulty in diagnosing and treating middle-ear infections and even created another surgical problem—that of treating middle-ear infections that had been badly mismanaged because of unwise reliance on the antibiotics, particularly penicillin, alone. It was forgotten that when suppuration began, antibiotics were of no avail, since the pathology is localized in its bony cage.

Another facet of this problem was that an apparently arrested otitis media recurred again and again, and became transformed into a chronic condition which injured hearing. The use of antibiotics masked the underlying condition by eliminating pain and fever, so that proper diagnosis was often missed.

It can be said now, I think, that otologists and general practitioners and pediatricians are coming back to the understanding that myringotomy remains a useful and necessary operation, in conjunction with antibiotic therapy.

The oversimplification of the problem of treatment of chronic ear infections with antibiotics had another disadvantageous aspect. That was its effect on interest in otology as an important field of specialization. For a time, in the 1940's, it became popular in medical circles to speak of otology as a dead or dying specialty. Fewer men were undertaking training in otology; hospitals had difficulty in obtaining the requisite number of residents in this specialty, and fewer well-trained ear surgeons undertook the practice of otology.

Now, evidently the perspective is better, and a new crop of men has been attracted to otology, perhaps by the popularization of the revived stapes mobilization technique. Whatever the reason, it is hoped that the talented among them will stay in the field, will explore those areas in which so much fundamental knowledge and endeavor remain to be acquired, and in which so much basic theoretical and clinical research remains to be done. If they will obtain the intensive training and experience that is required of competent temporal bone surgeons, they will find their work highly rewarding and will make real contributions to the cause of good medical practice and to the community.

Tympanoplasty and Related Procedures

In the current interest in tympanoplasty we again see a reversion to an earlier period in the evolutionary development of temporal bone surgery.

Politzer reported on early attempts by several 19th century otologists to close tympanic membrane perforations with skin grafts. Evidently the trials were not successful, and like early stapes mobilization

attempts, were soon abandoned. Kessel, in Europe, and Roosa in the United States, are among those whom Politzer mentions in his "History of Otology" as having tried plastic repair of the drum membrane without success.

Then, as now, the efforts at plastic closure of perforations in the eardrum were directed toward (1) removal of pathology, and (2) reconstruction of what in effect would be a new middle ear, which would constitute a closed, air-filled space lined with mucosa and which would communicate with the nasopharynx, that is, with an open Eustachian tube and sealed-off round window membrane.

In Europe, on the basis of the principle of surgical remobilization of the inner-ear fluids, Wullstein and Zöllner have contributed greatly towards the improvement of tympanoplasty technique. In the United States a number of temporal bone surgeons are making attempts to employ this surgery and to devise their own modifications of it.

Another and, to my mind, a more logical surgical procedure for the improvement of hearing in deafness secondary to chronic middle-ear suppuration has been introduced by J. H. Thomas Rambo. It is a combination of fenestration and tympanoplasty and is termed "musculoplasty." The viable temporal muscle pedicle flap is used to cover the new fenestra and to create a tympanic air-space. The continuous maintenance of the initial improvements in hearing obtained with this surgery will ultimately depend upon the amount of consideration given to the biological, physiological, and physical factors involved.

The basic principle upon which today's surgery is based—under the name of tympanoplasty—is still the remobilization of the inner-ear fluids. Only time will tell whether the long-term results of these renewed efforts will be any better.

The Reintroduction of Miot's Stapes Mobilization Technique

The recent reintroduction of Miot's stapes mobilization technique raises some interesting historical questions. It is approximately 60 years since he reported on 126 cases, with indicated success in 74. Significantly the literature of the day offers little evidence that the technique was successfully used by any other of the many great temporal bone surgeons of that era. In fact the literature discloses fully that stapes mobilization attempts were so rarely successful that the procedure was immediately made more drastic. Stapedectomy as a trial-and-error procedure in stapes ankyloses came quickly on the heels of the attempts to mobilize the rigid stapes.

It does not seem reasonable to assume that the great surgeons of that period who were constantly seeking a method of surgical intervention for the improvement of hearing in stapes fixation would have abandoned the use of a technique which initially seemed to offer so much. Fenestration of the semicircular canal had not yet been thought of and, indeed, stapes mobilization was the only method which had been suggested by anyone. Given a group of outstanding ear surgeons and any technique for possible improvement of hearing, is it not logical to assume that if the procedure offered even limited success, it would have been tried? Yet there is no mention in the literature of the day that Politzer, or Neumann, or Leidler, or any of the other great European otologists tried it. Blake, a Politzer pupil, made some few attempts to obtain hearing improvement with stapes mobilization, but he, too, quickly went on to the more drastic and inherently more uncertain procedure of removing the stapes. Finally the very dangerous operation of making a hole in the footplate was tried.

As of this writing, no one has attempted to explain this historical anomaly. Although there are many accounts of the clinical success of the procedure since its reintroduction five years ago, there is yet a complete dearth of convincing evidence which would justify its use on fundamental surgical principles.

Surgical intervention in deafness of otosclerotic origin by stapes mobilization, or

any variation of the technique, should rest on the following two principles: (1) the ability, preoperatively, to diagnose the possibility of successfully mobilizing the stapediovestibular joint without disruption of the ossicular chain, and (2) the possibility of deliberately providing for the permanent maintenance of the mobilized state of the stapes. Until evidence is forthcoming that these basic conditions are satisfied, the Miot technique will not have earned its place as an acceptable procedure in the surgery for otosclerotic deafness.

Mobilization of the stapes, as practiced today, is a destructive, nonsurgical orthopedic procedure, with complete disregard for the biological and physiological factors upon which maintenance of the improvement will depend. Because the mobilization is carried out in the immediate region of an actively growing tumor, the fixation is almost certain to recur.

The assurances that, if mobilization fails, fenestration may then be undertaken, is a basic oversimplification of the problem. However, to the extent that those who submit to the stapedial mobilization technique rely on the dubious promise that if it fails they can always resort to the time-proved fenestration operation, it is the greatest possible tribute to the Lempert fenestra nov-ovalis operation. Although these assurances are, in many instances, doubtful of accomplishment, they may well account for the current acceptance of the mobilization procedure.

119 E. 74th St. (21).

BIBLIOGRAPHY

For Early History

Ballance, C. A.: Surgery of the Temporal Bone, New York, The Macmillan Company, 1920, Vols. 1 and 2.

Kopetzky, S. J.: Otologic Surgery, New York, Paul B. Hoeber, Inc., 1925.

Otosclerosis: A Résumé of the Literature to July, 1928, edited by A. B. Duel, Committee on Otosclerosis, American Otological Society, New York, Paul B. Hoeber, Inc., 1929, Vols. 1 and 2.

Otosclerosis: A Résumé of the Literature—1928-1935, edited by A. B. Duel and E. P. Fowler, Central Bureau of Research, American Otological Society, St. Louis, Annals Publishing Company, 1936, Vol. 3.

Politzer, A.: Geschichte der Ohrenheilkunde, Stuttgart, Ferdinand Enke, Vol. 1, 1907 and Vol. 2, 1913.

Stevenson, R. S., and Guthrie, D.: A History of Oto-Laryngology, Ed. 2, Edinburgh, E. & S. Livingstone, 1949.

Whiting, F.: The Modern Mastoid Operation, Philadelphia, P. Blakiston's Son & Co., 1905.

For the 20th Century

Almour, R.: Surgery of Suppurative Petrositis, in Surgery of the Ear, edited by S. J. Kopetzky, New York, Thos. Nelson & Sons, 1947, Chap. 11.

von Békésy, G.: J. Acoust. Soc. America 21:245 (May) 1949, and subseq.

New Audiometer, Acta oto-laryng. 35:411, 1947.

Boucheron, E.: Mobilisation de l'étrier, Bull. méd. Paris 2:1225, 1888; Compt. rend. cong. Internat. Paris, 1889, p. 950.

Dandy, W. E.: Surgery of Ménière's Disease, in Surgery of the Ear, edited by S. J. Kopetzky, New York, Thos. Nelson & Sons, 1947, Chap. 16.

Lempert, J.: Simple Subcortical Mastoidectomy, Arch. Otolaryng. 7:201 (March) 1928.

Radical Subcortical Mastoidectomy, Ann. mal. oreille 2:1 (Feb.) 1922.

Complete Apicectomy, New York J. Med. 36:17 (Sept.) 1936.

Endaural, Antauricular Surgical Approach to the Temporal Bone, Arch. Otolaryng. 27:555 (May) 1938.

Improvement of Hearing in Cases of Otosclerosis, A New, One Stage Surgical Technic, Arch. Otolaryng. 28:42 (July) 1938.

Bone-Dust-Free Lempert Fenestra Nov-Ovalis, Arch. Otolaryng. 47:280 (March) 1948.

Lempert Endaural Subcortical Mastoidotympanectomy for the Cure of Chronic Persistent Suppurative Otitis Media, Arch. Otolaryng. 49:20 (Jan.) 1949.

The Permanently Patent Fenestra Nov-Ovalis, Laryngoscope 61:215, (March) 1951.

Surgical Remobilization of the Inner Ear Fluids for the Improvement of Hearing in Middle Ear Deafness, Tr. Am. Acad. Ophth. 59:166 (March-April) 1955.

The Dry Physiologically Perfected Fenestra Nov-Ovalis Technique, A. M. A. Arch. Otolaryng. 66:35 (July) 1957.

——Wolff, D., et al.: New Theory for the Correlation of the Pathology and Symptomatology of Ménière's Disease, Ann. Otol. Rhin. & Laryng. 61:717 (Sept.) 1952.

Miot, C.: De la mobilisation de l'étrier, Rev. laryng. 10:49, 83, 113, 1890.

Moure, E. J.: De la mobilisation de l'étrier, Rev. laryng. 7:225 (April) 1880.

Otosclerosis: An Index of the Literature, edited by E. P. Fowler, The Central Bureau of Research of the American Otological Society, Vol. IV, 1936-1955.

Rambo, J. H. T.: A New Operation to Restore Hearing in Conductive Deafness of Chronic Suppurative Origin, A. M. A. Arch. Otolaryng. 66:525 (Nov.) 1957.

Musculoplasty, a New Operation for Suppurative Middle Ear Deafness, Tr. Am. Acad. Ophth. 62:166 (March-April) 1958.

Sourdille, M.: New Technique in the Surgical Treatment of Severe and Progressive Deafness from Otosclerosis, Bull. New York Acad. Med. 13:673 (Dec.) 1937.

Wever, E. G., and Lawrence, M.: Physiological Acoustics, Princeton, N. J., Princeton University Press, 1954.

Wullstein, H.: The Restoration of the Function of the Middle Ear in Chronic Otitis Media, Ann. Otol. Rhin. & Laryng. 65:1020 (Dec.) 1956.

Zöllner, F.: The Principles of Plastic Surgery of the Sound-Conducting Apparatus, J. Laryng. & Otol. 69:637 (Oct.) 1955.

Functional Microsurgical Procedure in Middle Ear for Chronic Purulent Otitis

Personal Technique

ENRICO BOZZI, M.D., Milan, Italy

Definition and Indications

The personal operative method we illustrate is indicated in chronic inflammatory processes of the middle ear, characterized by a perforated eardrum in the epitympanic posterior region; a limited and moderately active osteitis of the "bridge" or the attic can be present; absence of cholesteatoma and integrity of the ossicular chain are necessary.

In such cases the hearing loss can be quite considerable, despite the lack of objective findings in the ear; this is due to the limitation of movements of the ossicular chain, because of articular ankylosis or blockage with scarry granulation tissue secondary to an osteitic process.

The operative procedure we are illustrating permits access to the tympanic cavity and atticotomy; the tympanic cavity is reached by the retroauricular way, after the drum is detached, thus maintaining its preoperative condition. I apply the same technique as in functional surgical procedures for adhesive otitis, with the difference that in the former, because the pathology shows osteitic foci in the attic or the "bridge," we go ahead and remove these foci.

This procedure unables us to explore and attack surgically the following parts:

(*a*) The tympanic cavity. We can control extensively the functional efficiency of the anatomical elements and restore their function through the known principles of tympanoplasty and surgery of the oval window.

(*b*) The attic. We remove the osteitic foci, control the integrity of the ossicular chain, and particularly the efficiency of the incudomalleolar joint, restoring its efficiency with the same procedure used for functional surgery in adhesive otitis.

Submitted for publication June 16, 1958.

Physiopathology of Chronic Purulent Middle-Ear Otitis with Epitympanic Perforation

In the physiopathologic study of these lesions, we shall deal only with the typical situations involving the membrane in the epitympanic region and the attic. We deliberately discard all the other situations secondary to the anatomopathological conditions of the attic that can occur in the tympanic cavity and the adjacent structures. This is justified by the fact that in chronic purulent epitympanic otitis the lesions of the attic are essential; the lesions of the tympanic cavity are not indispensable and anyway they do not constitute a typical feature of the case we are examining; in this case we refer the reader to our extensive report at the 43d Congress of the Italian Society of Otology, 1955, the subject of which was "Functional Surgery of the Middle Ear."

The various possibilities are the following:

1. *The "pars flaccida" of the membrane is perforated.*

Shown in Figure 1, the most unfortunate occurrence for hearing loss is the free communication between the auditory meatus and the tympanic cavity, through the epitympanic perforation. This allows the sound waves, instead of following the ossicular chain, to go into the tympanic cavity, as indicated by arrow *A*, thus interfering with

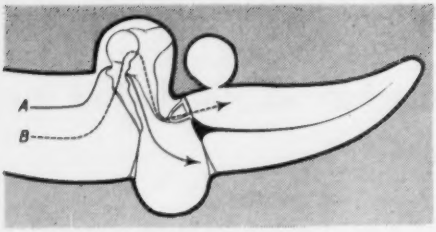


Fig. 1.—Chronic purulent epitympanic otitis media. The perforation of the "pars flaccida" of the drum allows the direct passage of the sound waves into the tympanic cavity.

the proper function of the windows. The arrows (the dotted one and the solid one) clearly indicate how this interference occurs: the sound waves, arriving directly through the perforation and stimulating the round window, oppose the physiologic stimulation of the ossicles, with the resulting partial exclusion of the stimuli arriving at the windows and consequently diminished sensitivity; this gives an impaired hearing of middle-ear origin with an audiometric curve resulting from the following conditions: (*a*) diminished functional surface of the membrane (due to the perforation which renders one portion unusable), with a relatively reduced sound pressure effectively selected by the membrane itself; this factor is much less important here than when the perforations are localized in the "pars tensa" of the membrane; (*b*) absence of the air cushion normally present in the tympanic cavity, which acts as a defense and equilibrium media; (*c*) probable uncoordination of the oscillatory phases of the different sections of the membrane.

It is evident that the damage occurring from an epitympanic lesion involves an area submitted to minimal excursions during vibration, and therefore the functional damage resulting from the perforation is minimal. We shall consequently have an improving air-conduction audiogram and the bone-conduction curve will be excellent, at least while the cochlear function is good.

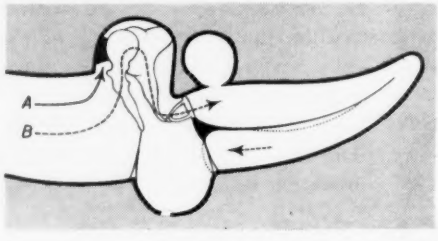
2. The inflammatory granulation tissue of the epitympanic region obstructs the perforation of the "pars flaccida."

In the anatomopathological picture schematically presented in Figure 2, the main lesions are the perforation of the drum and the presence of inflammatory granulation tissue that joins the head of the malleus to the epitympanum. The perforated drum causes limited functional impairment, as we previously mentioned, and this is due to the fact that the sound reaches the round window directly. If the localization of the granulation tissue gave a "pouch-like" perforation, without any communication with the tympanic cavity, the functional picture would undoubtedly be less compromised, because, despite the mechanical obstacle to the movements of the malleus, there would not be the inconvenience of the sound wave reaching the round window directly. This possibility is illustrated in Figure 2.

The presence of the granulation tissue joining the walls and the head of the malleus determines a diminished mobility of the malleus, with an increased rigidity of the ossicles and with an increased vibrating mass, resulting from the added granulation tissue. Because of the pouch-like perforation the functional picture does not reveal the disadvantage of the perforation, but shows clearly the inconvenience created by the additional mass, with an impairment in relation to the extent of the proliferative process, its density, and its topographical localization.

The air-conduction curve might be slightly decreasing (mass curve), with a good or excellent bone-conduction curve.

Fig. 2.—Chronic purulent epitympanic otitis media. There is no communication with the tympanic cavity through the perforation of the "pars flaccida" because of the presence of inflammatory granulation tissue in the epitympanic area.



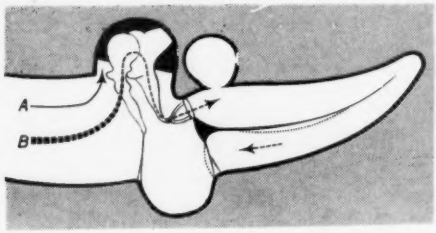


Fig. 3.—Chronic purulent epitympanic otitis media. The abundant granulation tissue of the epitympanum immobilizes the ossicles.

3. *The epitympanic granulation tissue interferes with the mobility of the ossicles.*

In Figure 3, we see an anatomopathological picture similar to that in Figure 2. Here, too, the inflammatory and adhesive process in the epitympanum creates an obstacle to the mobility of the ossicles, with relative impairment, with the difference that the granulation tissue in this case involves almost all the head of the malleus and the body of the incus and the short process. The dotted arrow illustrates the diminished sound wave arriving at the oval window through such an altered ossicle chain.

The diminished mobility of the ossicles will be quite noticeable, and the audiometric curve will show an increase due to the increased mass; the tympanic perforation in this case has been pouched by the granulation tissue; the presence of the granulation tissue is a useful occurrence, because the sound wave does not reach the tympanic cavity. Obviously this fact does not compensate for the much greater loss due to a diffuse granulation tissue, which causes a notable hearing loss.

Operative Procedure

We rely on the illustrations (Fig. 4 *A, B, C, D, E, F, G,*) to show more clearly and concisely the operative technique we have used.

With this surgical procedure we have attempted to find a solution to the physiopathological conditions of middle-ear epitympanic otitis, with the following steps: (1) freeing of the ossicles; (2) closure of the perforated drum; the outcome of the perforation is varied; if it is small, it comes to a spontaneous healing, otherwise we resort later to one of the various methods suggested to obtain the closure of the drum; (3) elimination of the suppurative process; this is essential for the ossicles to remain free in the future and for the drum to heal. The success of our technique is possible only with the help of antibiotics and the extensive and prolonged use of cortisone.

Medication

1. *During Operation.*—At the end of the procedure and with the cavity exposed, we fill it with a suspension of prednisolone (25 mg/cc.) and a concentrated antibiotic solution. After suturing the retroauricular incision we fill the auditory meatus with gelatin sponge (Spongostan) to avoid traumatizing the drum and to obtain better adhesion of the auditory meatus and the drum to the underlying structures.

2. *After Operation.*—Regular antibiotic treatment is continued. Early roentgentherapy is given with anti-inflammatory dosage. Within 7 days we remove the Spongostan from the auditory meatus, and 15 days later, with a very thin needle, we start the introduction into the attic, through the upper wall of the auditory meatus, of a prednisolone suspension (25 mg/cc.). This therapy will be repeated twice weekly for 15 days and then weekly for a 3-4 month period; at this time one presumes the inflammatory processes have subsided and recurrent adhesions are excluded.

Advantages of This Method

This method has significant advantages. The drum is not involved in the operation. In order to explore the tympanic cavity, the drum is not detached through the auditory meatus, as in Lempert's or Rosen's method (which would easily cause an enlargement of the perforation which would not be spontaneously or easily repaired). It is also unnecessary to remove the drum and substitute it later by a surgical procedure.

The visualization of the tympanic cavity is quite adequate for the inspection of the

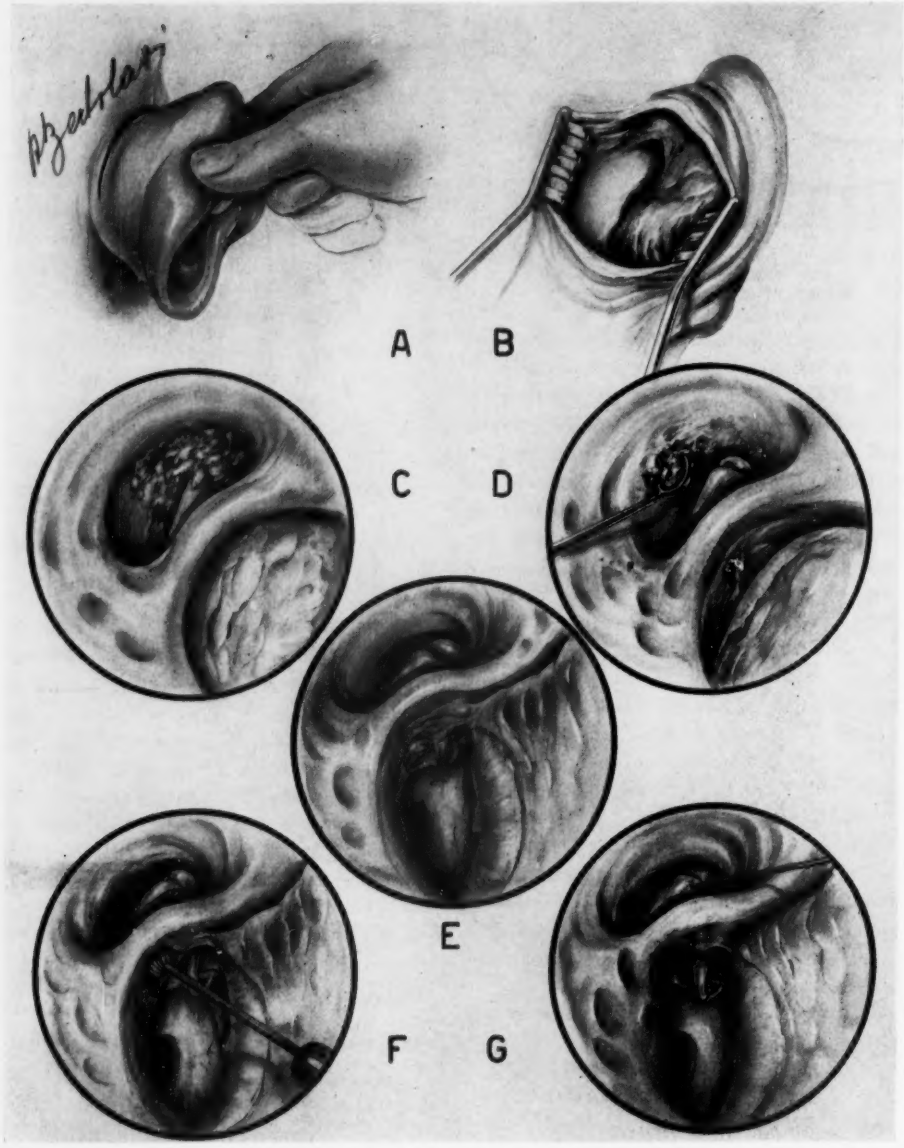


Fig. 4.—*A*, incision of the soft tissues on the supra- and retroauricular groove, extended from the root of the helix to a transversal line passing by the inferior margin of the auditory meatus. *B*, detaching and lifting of the temporal muscle; exposure of the posterior and superior limit of the auditory meatus. *C*, separation of the skin covering the posterior and superior portion of the bony auditory meatus, up to the tympanic membrane. Opening of the attic, with exposure of the incus and the head of the malleus incorporated in a scarry and inflammatory tissue; partial removal of the posterior and superior wall of the bony auditory meatus, leaving a bridge between the attic and the tympanic cavity, 1 cm. high. *D*, exposure of the ossicle and especially the incudomalleolar joint, with careful removal of the pathologic tissues filling the attic. *E*, after detaching the tympanic membrane along a line going from 7 to 12 on a clock-dial, we notice (1) a small osteitic focus located on the anterior and inferior wall

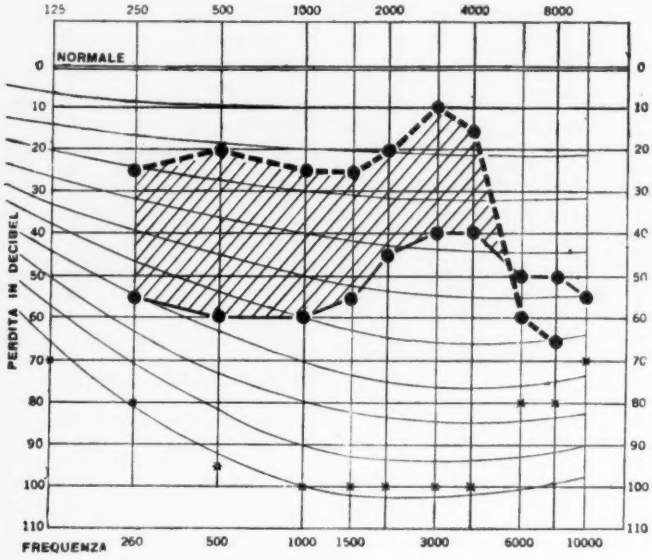


Fig. 5 (Case 1).—Functional hearing benefit 10 days postoperatively.

different elements and for any eventual surgical treatment (mobilization of the stapes). The attic is exposed entirely and this permits removal of any osteitic focus and manipulation to free and mobilize the incudomalleolar joint. In case it is not possible to mobilize the stapes in the oval window or the ossicles, because of excessive adhesions or ossification of the single elements of the ossicle chain, it will always be possible, instead of following the previous operative plan, to proceed to do a fenestration of the lateral semicircular canal. This fenestration will be undertaken in two stages (following Rüedi) to avoid infecting the labyrinth.

If we are suddenly confronted with an interruption of the ossicular chain, despite the audiograms, stratigraphic examinations, and acoustic probe, it will be easy to resort to one of the different tympanoplastic techniques, according to the anatomopathological condition.

Report of Cases

CASE 1.—A 25-year-old man has right otorrhea of three years' duration, periodic, without foul odor. He complains of a marked hearing defect on the right side; the exact time of onset is unknown, but it has remained stationary for a long while; slight headache, localized in the right temporal region, of a few months' duration. Physical examination of the right ear shows a dry auditory meatus; the tympanic membrane is totally dull, thickened, slightly retracted. In the attic in the posterior-superior region, one can see a perforation filled with a drop of mucopurulent secretion.

Salpingoscopy revealed no pathological findings. The audiograms show a marked transmission defect on the right; cochlea and eighth nerve are intact; recruitment negative. The roentgenograms show a poor pneumatization of both mastoids, while no bone destructive lesions are seen in the attic or the ossicles.

of the "bridge," near the facial nerve elbow, corresponding to the perforation; (2) inflammatory tissue covering totally the long process of the incus, the stapes, and the oval window, loosely adherent to the internal surface of the tympanic membrane; (3) small, thick mucopurulent secretion, limited to the oval window, while the tympanic cavity is free; (4) free round window and promontory; with a probe one can explore the patency of the tube; (5) the stapes is mobilized by exerting a pressure with a probe and the membrane of the round window responds accordingly. *F*, the ossicles are now exposed after removal of the inflammatory tissue surrounding the stapes, the long process of the incus, the incudostapedial joint and the oval window. Removal with a fraise of an osteitic focus on the "bridge." *G*, we test the normal mobility of the joints of the ossicle chain with a probe, after removing all pathological processes from the cavity and exposing the single elements of the attic and the tympanic cavity.

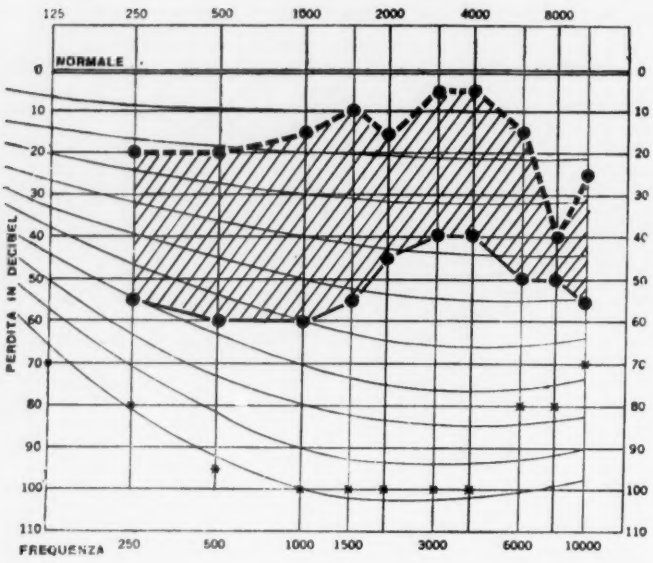


Fig. 6 (Case 1).—Five months after operation, following cortisone treatment, the functional hearing benefit is even greater than that shown in Figure 1.

Anatomopathological Findings and Surgical Procedure.—Upon exploration of the attic we notice abundant inflammatory granulation tissue that fills this space almost entirely, mixed with slight mucus covering the ossicles; integrity of the ossicular chain.

The exploration of the tympanic cavity shows the following: (*a*) a small osteitic focus localized on the anterior and posterior wall of the "bridge," at the level of the perforation of the tympanic membrane; (*b*) a granulation tissue, of jelly-like consistency, covering totally the long process of the incus and the stapes, and adherent to the internal side of the tympanic membrane, in its portion next to the "bridge"; (*c*) slight dense mucus among these granulations; (*d*) round window, promontory, and Eustachian tube are normal; the stapes is mobile, and upon pressure we discern a visible movement of the secondary tympanum.

We remove the granulation tissue surrounding the ossicles, likewise we remove with the bur the osteitic focus described on the "bridge"; with a small probe we make sure of the mobility of the stapes and the incudomalleolar joint. The audiogram, 10 days after operation (Fig. 5) shows the remarkable functional hearing gain.

Fifteen days after the procedure, we introduce into the attic a concentrated cortisone suspension, at first twice, then once weekly.

The audiogram five months after operation (Fig. 6) shows the persistence of the hearing gain. The perforation of the tympanic membrane has closed rapidly and after a while one cannot recognize its location.

CASE 2.—Girl 18 years old. Continuous right otorrhea since childhood; the discharge has increased in the past months, and pain in the right mastoid with headache have appeared. Upon physical examination, the right tympanic membrane is injected and thickened; in the epitympanic region one sees a fairly large perforation with clear-cut borders, discharging a slight amount of pus.

The audiogram is typical of the pure transmission type; moderate impairment; roentgenography not demonstrative of any lesion.

Anatomopathological Report and Operative Procedure.—On opening the attic, the malleus and incus are found surrounded by scarcely adherent granulations; no pus or osteitis; the incudomalleolar joint is free. Upon exploration of the tympanic cavity, one notes granulation tissue involving the long process of the incus and stapes, which does not appear mobile; these inflammatory tissues reach under the "bridge" and in the space between incus and body of the malleus. Round window, promontory, and Eustachian orifice are normal.

After detaching the tympanic membrane, we free the long process of the incus and stapes from the granulation tissue surrounding them. Mobilization of the stapes. The ossicles of the attic are protected with an amniotic membrane and an absorbable jelly. Here again we instill cortisone in the attic; because of the patient's neglect, the treatment is interrupted after 20 days. A month after operation, the clinical recovery is complete, and the tympanic perforation is closed. The audio-

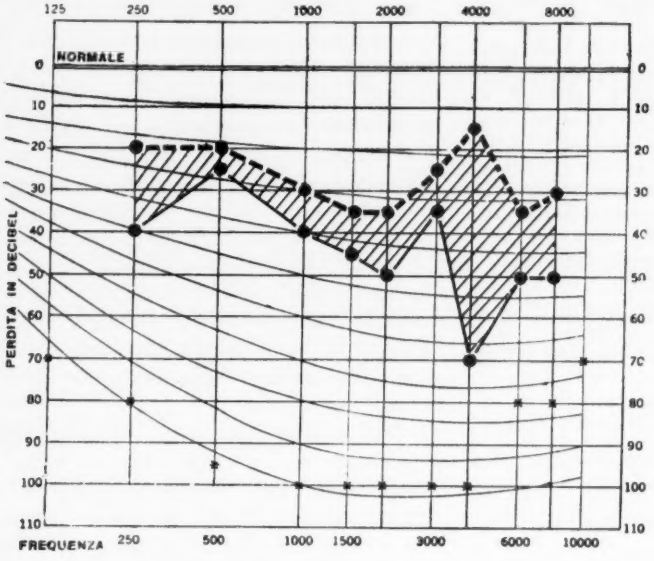


Fig. 7 (Case 2).—Functional hearing benefit three months after operation.

gram, three months later (Fig. 7) shows the functional benefit obtained.

CASE 3.—A 48-year-old man. No particular complaint, except a persistent bilateral ear discharge of many years' duration and a pure-tone transmission hearing deficit of 30%, bilateral and equal. Upon physical examination, the right tympanic membrane is found thickened and retracted, with a small perforation slightly discharging in the epitympanic posterior area; the left drum has a large perforation in the posterior inferior section, with an abundant, but not foul-smelling, discharge.

Anatomopathological Report and Surgical Procedure.—After the attic is opened and the granulation tissue covering the ossicles is removed, their integrity is controlled; the incudomalleolar joint is mobile and it is exposed by mistake during operation.

After detaching the drum, we can explore the tympanic cavity. An osteitic focus on the anterior

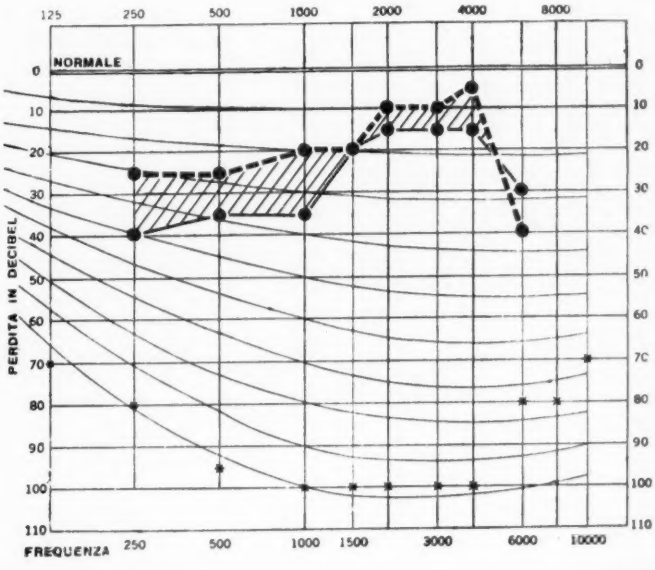


Fig. 8 (Case 3).—Functional hearing benefit four months after treatment.

border of the "bridge" is removed. Abundant inflammatory granulation tissue covers the long process of the incus and the stapes, along with a slight mucopurulent secretion; they are removed, and the normal mobility of the stapes is controlled. The tip of the long process of the incus is slightly eroded and compromises the continuity of the ossicular chain; the long process is cleaned and placed in contact with the head of the incus. The round window and the Eustachian orifice are free.

Twenty days after this procedure, because of the persistence of the epitympanic perforation, we undertake a total skin myringoplasty. We continue weekly to introduce cortisone suspensions in the attic. The audiogram (Fig. 8) shows the functional gain achieved four months after the procedure.

Comment on the Clinical Cases

In all cases we have limited bone destruction to the minimum necessary; we have removed the granulation tissue and have respected (and even reconstructed in the third case) the anatomical elements that are necessary for the perfect efficiency of the middle ear.

After operation, the purulent process of the middle ear has always been cured rapidly and completely.

Follow-up showed the hearing gain was excellent in Case 1, already noticeable on the 10th postoperative day; it increased within a month and thereafter remained unchanged.

In Case 2, we obtained a good functional result, despite the fact that postoperative instillation of cortisone could be done only five times.

In Case 3, there was moderate hearing benefit; this partial result can be explained, I believe, by the interruption of the ossicular chain, caused by caries of the incus, which could not be adequately repaired.

The systemic and prolonged introduction of cortisone derivatives in the operative cavity (attic and tympanic cavity) certainly prevents the formation of adhesions that would endanger the future function of the ear which has been subjected to operation.

Summary and Conclusions

A method is presented which is indicated in the chronic inflammatory lesions of the middle ear, characterized by a posterior epitympanic perforation and by a moderate and limited osteitis of the "bridge" and the attic; absence of cholesteatoma and integrity of the ossicles are necessary. In such cases the hearing damage can be considerable, despite the minimal physical findings; this is due to the limited movements in the ossicle chain, caused by ankylosis of the joint and blockage with scarry and inflammatory tissue, secondary to the osteitic lesion.

This method consists of a combination of procedures: (*a*) exploration of the tympanic cavity after detachment of the drum by the retromandibular approach; (*b*) an atticotomy, with final removal of the small osteitic focuses of the attic or the "bridge."

The damage to the membrane is minimal, and a possible enlargement of the previous perforation is avoided.

This procedure permits us to explore and attack surgically (*a*) the tympanic cavity: we control extensively the functional efficiency of the anatomical elements, and we restore their efficiency with the aid of the well-known basic elements of tympanoplasty and surgery of the oval window; (*b*) the attic: we remove the osteitic foci, we control the integrity of the ossicular chain, and especially the efficiency of the incudomalleolar joint, restoring its function with the same procedure we used for functional surgery in adhesive otitis.

In case, during operation, we come across anatomopathological lesions more serious than the ones expected for the indicative procedure, it will be easy to resort to a fenestration of the lateral semicircular canal (to be performed in two steps, according to Rüedi's technique) or to one of the varied tympanoplastic procedures, according to the anatomopathological conditions.

Piazza Repubblica 27.

Some Observations on the Pathogenesis of Middle-Ear Cholesteatoma

CÉSAR FERNÁNDEZ, M.D.; JOHN R. LINDSAY, M.D., and MERLE MOSKOWITZ, M.D., Chicago

The widespread use of chemotherapy and antibiotics has modified considerably the general picture of middle-ear cholesteatoma.

Cholesteatoma which originates by immigration of epidermis through a marginal or total perforation of pars tensa after necrotizing otitis media, as substantiated by Habermann [1] and Polvogt,[2] has been greatly reduced, while the incidence of attic cholesteatoma has shown no definite decrease.

The origin of attic cholesteatoma has not been agreed upon in spite of extensive clinical and histopathologic studies. The evidence presented by various sources supports the theory of several origins. No single explanation has been adequate to account for all types which have been examined histologically or observed clinically.

The current theories may be summarized as follows:

1. Epidermoid theory. This theory assumes the inclusion during embryonic life of fetal epidermal cells. McKenzie [3] argued that all middle-ear cholesteatomas are primarily epidermoids of the same nature as the intracranial cholesteatoma of Johannes Müller.[4] McKenzie believed that the tumor-like growth could expand considerably into the attic, antrum, and mastoid without infection. It might perforate Shrapnell's membrane and evacuate the contents, in which case the ingrowth might stop, but more frequently the cholesteatoma becomes infected, giving rise to a chronic otitis media.

Diamant [5] pointed out the question of chronological order or cause-effect relation between cholesteatoma and chronic otitis media with marginal perforation. In his theory, the initial condition is the presence of a "cholesteatoma germ" in the epithelium of the middle ear. In some cases it is conceivable that development of the cholesteatoma germ into a cholesteatoma is the primary phenomenon and the marginal perforation is secondary. In other cases the conditions may be different. Diamant clearly stated that the assumption of cholesteatoma germ in the middle ear mucosa "does not necessarily involve assenting to the belief that cholesteatoma is a genuine tumor."

Teed [6] agreed that those cases of "primary cholesteatoma of middle ear and the mastoid may result from the anomalous functioning of normally placed epidermal cells."

The extensive reviews of Holmes [7] and Nager [8] of a large series of middle-ear cholesteatomas failed to demonstrate a single case of congenital origin.

It is accepted that the congenital cholesteatoma is so rare that its incidence is unimportant for practical purposes. Nevertheless, the existence of such cases seems established.

2. Traumatic theory. Cholesteatoma which developed after trauma—either accidental or surgical—involving the middle ear has been reported several times.[9-12] The relationship between trauma and attic cholesteatoma seems to be well established although its incidence is small.

Submitted for publication Aug. 15, 1958.

Supported by the Central Bureau of Research of the American Otological Society.

From the Division of Otolaryngology of the University of Chicago. From the Department of Pathology of the University of Chicago (Dr. Moskowitz).

3. Metaplasia theory. This theory has been sustained lately by Tumarkin.[13] His argument is that the hypotympanum has a ciliated epithelium while the epitympanum is squamous. During infection, the former gives rise to a mucopurulent otitis media; the latter tends to proliferate and form squames, and consequently a cholesteatoma may develop. The origin, however, lacks the support of convincing histopathologic evidence.

4. Wittmaack's theory.[14] The necessary conditions are (*a*) Prussak's space and attic form an independent air-containing cavity which is secluded from the hypotympanum by bridges of organized tissue. These are sequelae of an asymptomatic otitis in early life. (*b*) The mucosa of this cavity remains hyperplastic, which implies the presence of a dense vascular supply. The end-result of the first condition is the absorption of air, creating a negative pressure in the closed space, and consequently Shrapnell's membrane is drawn into Prussak's space and attic. The second condition—the biological factor—stimulates the ingrowth of the epidermis. In the pouch, desquamation accumulates, giving rise to the cholesteatoma. This theory is no longer considered satisfactory.

5. Day's theory.[15] According to this author the initial condition is the inflammatory reaction of the loose, embryonic tissue of the middle ear in early life, sealing off the epitympanum from the tubotympanic cavity. Absorption of any air contained in the closed cavity creates a negative pressure and sinking of Shrapnell's membrane, which may remain in that position by adherence to the attic walls. The second—indispensable condition—is the presence of a perforation in Shrapnell's membrane through which epidermis immigrates. Finally, cholesteatoma will develop only if desquamation appears and the outlet for the debris is inadequate. The primary stimulus for desquamation is moisture—water, serum, mucus, or pus. In the absence of moisture, even a cholesteatoma may become dry and quiescent.

6. Ojala and Saxén's theory. In a well-documented paper[16] these authors demonstrated that "mere indrawing of Shrapnell's membrane without continued inflammatory irritation does not lead to attic cholesteatoma." The initial condition is the inflammatory process in the middle ear. If perforation occurs, then the epidermis may immigrate through it into the Prussak's space and/or attic, where it may follow two pathways: (*a*) Growth through granulation tissue or over the injured mucosa. Desquamation, accumulation of debris and inadequate drainage, lead to cholesteatoma. When the inflammatory process heals, probably further development of cholesteatoma stops. (*b*) Growth through the connective tissue of submucosa leading to a cholesteatoma as before. When the inflammation subsides, it may continue growing, provided the mucosa remains hyperplastic.

If the initial condition—the inflammatory process—does not lead to perforation of Shrapnell's membrane, cholesteatoma may also develop, provided the inflammation has produced clefts or openings in the corium. The corium of Shrapnell's membrane is a uniform, loose connective tissue which originates in the skin of the external canal and acts as a barrier for ingrowth of epidermis. Under favorable circumstances, the epidermis is stimulated by the inflammation and grows through the clefts or openings of the corium, thus invading Prussak's space and attic. Cornification and accumulation of debris in the ingrowth lead to cholesteatoma. Under some conditions, irritative lesions of the external canal may also act as stimulus for the ingrowth of the epidermis.

7. Rüedi's theory.[17] For this author the initial condition is an inflammatory process of the middle ear. Attic cholesteatoma may develop by immigration of epidermis through a perforation of Shrapnell's membrane or by ingrowth of epidermic cones from the basal cells. Rüedi stated that "immigration is possible by the normally inborn energy of growth which is present in early infancy in the epidermis of the

auditory canal adjoining the tympanic membrane superiorly. On the other hand, active growth of the cones of the basal cells is enhanced by submucosal connective tissue filling the incompletely pneumatized epitympanal spaces and cells."

It is apparent that the theories of Witmaack, Day, Ojala and Saxén, and Rüedi are all variations of the immigration theory. The last two offer an explanation for the relatively rare occurrence of a cholesteatoma in the attic without a perforation.

Clinical observation suggests strongly that there are two types which develop with an attic perforation: one in which the cholesteatoma is preceded by repeated acute inflammatory middle-ear episodes, and a second in which such a history is lacking but in which a cholesteatoma is found behind a small perforation in Shrapnell's membrane.

The first type fits the general description given by Day, Ojala and Saxén, and Rüedi with possibly some minor variations. One of these is that the hyperplastic tissue which occupies the attic and/or aditus contains young fibrous tissue. The contraction of this tissue produces retraction of the drum. This occurs usually during the quiescent period after the acute inflammation has subsided. Shrapnell's membrane is most susceptible because of its location and lack of a fibrous middle layer. The stage is thereby set for the formation of a cholesteatoma with the next acute inflammatory episode. Swelling, exudation, and exfoliation of the epidermis with retention in the retracted pocket may follow. If this debris is promptly removed, the cholesteatoma formation may be averted until a subsequent acute episode occurs.

The role which can be attributed to contraction of the fibrous tissue in the attic or aditus is better illustrated by certain cases in which the posterosuperior quadrant of the drum becomes retracted and is the starting point of the cholesteatoma. In these the process is similar but relatively less frequent. The mucosal lining as well as the dense fibrous middle layer of the drum are destroyed by the inflammatory process, and adhesions are formed. Contraction of the fibrous tissue causes retraction beneath the incus into the aditus, thereby predisposing to cholesteatoma, as in the case of the retracted Shrapnell's membrane. In the case of the membrana propria it is only when the fibrous middle layer is destroyed that cholesteatoma may develop. Ojala and Saxén claim that the corium prevents ingrowth of the basal-cell layer of Shrapnell's membrane and that a defect is necessary for cholesteatoma formation.

It appears, therefore, that contraction of fibrous tissue must be considered as a primary predisposing factor in the formation of this type of attic cholesteatoma.

A second type of attic cholesteatoma is that in which a history of recurring otitis media is lacking and signs of retraction of the membrana propria absent; the genesis of the cholesteatoma in this type of case has never been clearly established. The observations which we have made in a series of animals with suppurative otitis media provide some evidence as to how this type of cholesteatoma may develop. The findings to be described in the ears of these animals demonstrate clearly that the epidermis may immigrate into the epitympanum through a perforation either in Shrapnell's membrane or bordering on it and thus provide the basis for a cholesteatoma.

Material

The material to be presented here consists of histologic findings in the temporal bones of rats which were treated with estrogens for the purpose of studying changes in serum lipids and coronary arteries.[18]

All the animals were killed with ether anesthesia. After autopsy was completed, the temporal bones were removed in one block and immersed in Heidenhain-Susa solution. Subsequent treatment was as described elsewhere.[19] Serial sections were stained with hematoxylin-eosin or Mallory's azan and studied under regular light microscopy.

Report of Cases

CASE 1.—This animal presented a chronic otitis media and a purulent collection in the superior wall

Fig. 1.—Abscess in superior wall of external canal opening into attic and hypotympanum. (1) External canal. Tympanic membrane was not perforated. (2) Hypotympanum showing hyperplasia and cystic formation of the lining. The cavity filled with pus, which is draining through the Eustachian tube. (3) Part of the incus embedded in connective and inflammatory tissues. (4) Abscess extending from the attic. Shrapnell's membrane elevated. (5) Facial nerve. Hematoxylin-eosin stain; × 39.

of the external auditory canal. The abscess bordered on Shrapnell's membrane and the skin extending outward from it had apparently resulted from the middle-ear infection as a localized pocket of pus in the lateral part of the attic. The study of serial sections showed that the abscess cavity connected with the tympanum proper through a small channel. No perforation to the external canal was found. The photomicrograph of Figure 1 partially illustrates this finding. Shrapnell's membrane was elevated so that the abscess was a continuation of the attic. The epidermis of the external canal and Shrapnell's membrane showed neither tendency to ingrowth nor major change in its structure. Below the dermic layer a dense capsule of fibrous tissue was present, and the lining of the abscess cavity was formed by inflammatory tissue.

Comment.—This case illustrates a collection of pus filling the attic and extending beyond its margins beneath the skin. No perforation had occurred to the external canal. The capsule of fibrous tissue beneath the epidermis had resisted the action of the suppurative process probably because an outlet existed down into the tympanum.

Case 2.—This animal also presented a chronic otitis media and a purulent collection under the skin of the superior wall, including Shrapnell's membrane, but in this case the abscess was open to the external canal, as illustrated in the upper photomicrograph of Figure 2. Shrapnell's membrane was elevated. The lining of the abscess was formed by a thick layer of inflammatory tissue with numerous cystic formations. The ossicles were embedded in fibrous and inflammatory tissue. The perforation was in the skin adjacent to Shrapnell's membrane. The epidermis in the neighborhood of the stoma showed signs of irritation (hyperplasia) and had begun to immigrate into the abscess, as the lower photomicrograph of Figure 2 illustrates.

Comment.—The origin of the lesion seemed to be, as in Case 1, an expansion of the infection from the attic, formation of abscess under the skin of the superior

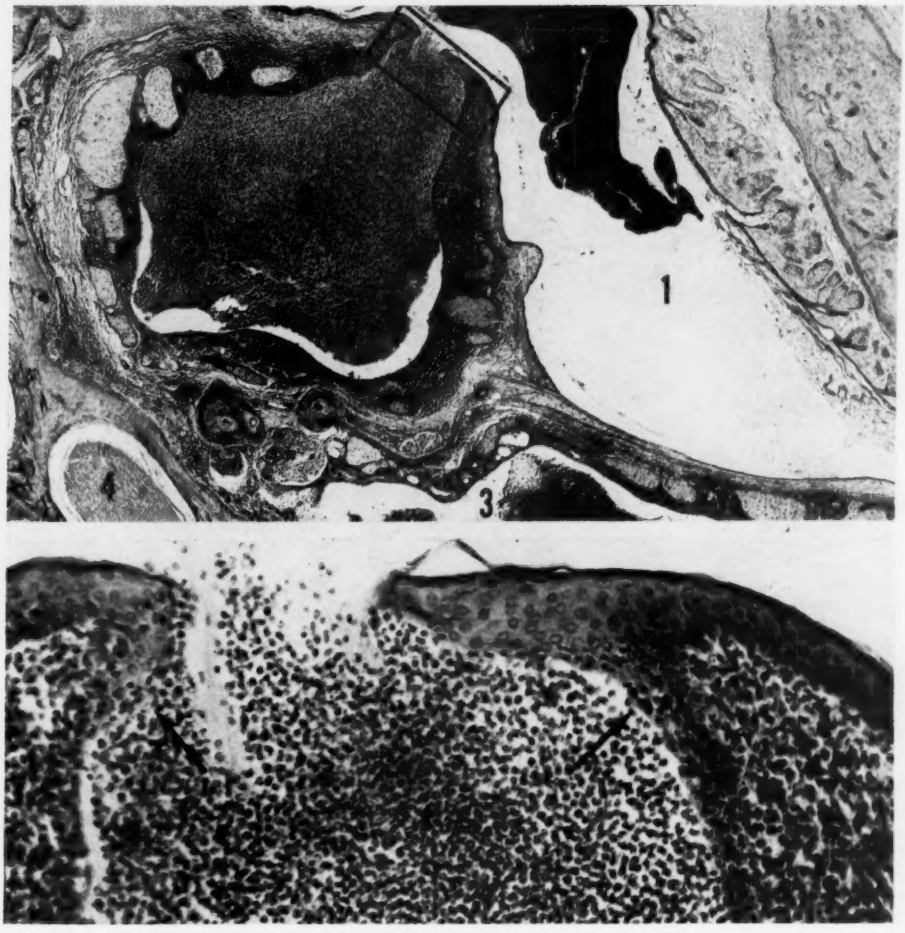


Fig. 2.—(Upper) Abscess extending from the attic with perforation into the external canal. (1) External canal with pus and desquamated debris. Membrana propria was intact. (2) Abscess, with opening in the skin of external canal adjacent to Shrapnell's membrane. Numerous cystic formations in the walls of abscess cavity. Ossicles embedded in connective and inflammatory tissue. (3) Hypotympanum showing hyperplasia and cystic formation of the lining. Cavity is filled with pus which drained through Eustachian tube. (4) Facial nerve. Hematoxylin-eosin stain; × 36.

(Lower) Higher magnification of the region enclosed in the rectangle of upper photomicrograph. At the stoma the skin shows first stage of immigration into the cavity as arrows indicate. × 245.

wall, and rupture into the external canal. The case illustrates the early stage of immigration of the epidermis through a perforation.

Case 3.—As in the previous two cases, this animal had a chronic otitis media and a localized collection of pus occupying the attic and the adjacent region. A perforation had occurred to the external canal. The stoma was at the boundary of the skin in Shrapnell's membrane. The photomicrograph of Figure 3 shows the immigration of skin on one side, whereas the epidermis of Shrapnell's membrane seems to form the opposite border of the perforation. The upper photomicrograph of Figure 4 shows the continuity of the skin on one side and epidermis of Shrapnell's membrane on the other with the lining of the cavity. Furthermore, the histologic types of the repairing epithelium in the cavity are similar to

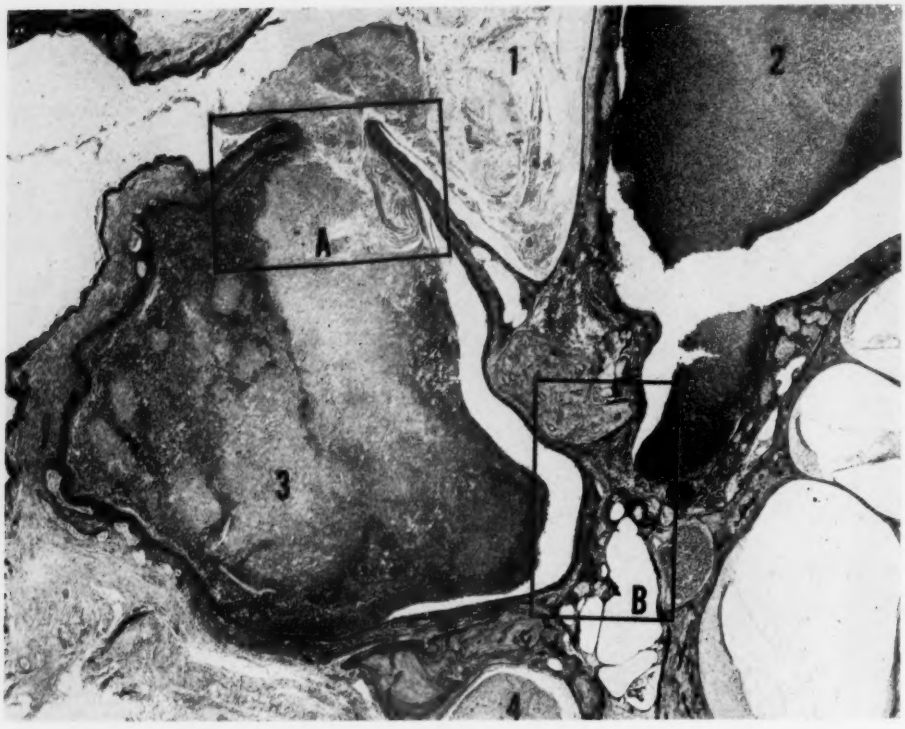


Fig. 3.—Abscess extending from the attic into the external canal. (1) External canal filled with pus, debris, and desquamated epithelium. Membrana propria not perforated. (2) Hypotympanum. Hyperplasia and cystic formation of the lining. (3) Abscess with stoma at the border between skin and Shrapnell's membrane. Lining of the cavity is formed by immigration of both skin and epidermis of Shrapnell's membrane. (4) Facial nerve. Regions enclosed in rectangles *A* and *B* are shown at higher magnification in Figure 4, *A* and *B* respectively. Hematoxylin-eosin stain; × 31.

the original (Figs. 5 and 6), although the basal cell layer of the lining corresponding to the skin is thicker. This indicates greater activity, probably due to irritation by the contents of the abscess. The immigrating epithelium lined the cavity completely except at the boundary of the attic, as shown by the lower photomicrograph of Figure 4. Pus, debris, and a collection of exfoliated cells occupied the cavity (Fig. 4, *A*) and external canal (Fig. 3).

Comment.—The origin of the abscess seemed to be the same as in previous cases. This temporal bone is particularly interesting because it definitely illustrates the immigration of skin and epidermis of Shrapnell's membrane through a perforation. Also the repairing epithelium is histologically of the same type as the original.

General Comment

The review of theories concerning the origin of attic cholesteatoma, seems to indicate that it may develop from several sources. A proved congenital origin corresponding to the true cholesteatoma of Müller is an extremely rare occurrence in the ear.

A traumatic origin has been demonstrated clinically and by histopathologic studies.

An origin by metaplasia, although theoretically conceivable, still lacks convincing clinical and histopathologic evidence.

The ingrowth of the epidermis into the attic has been demonstrated by many histo-

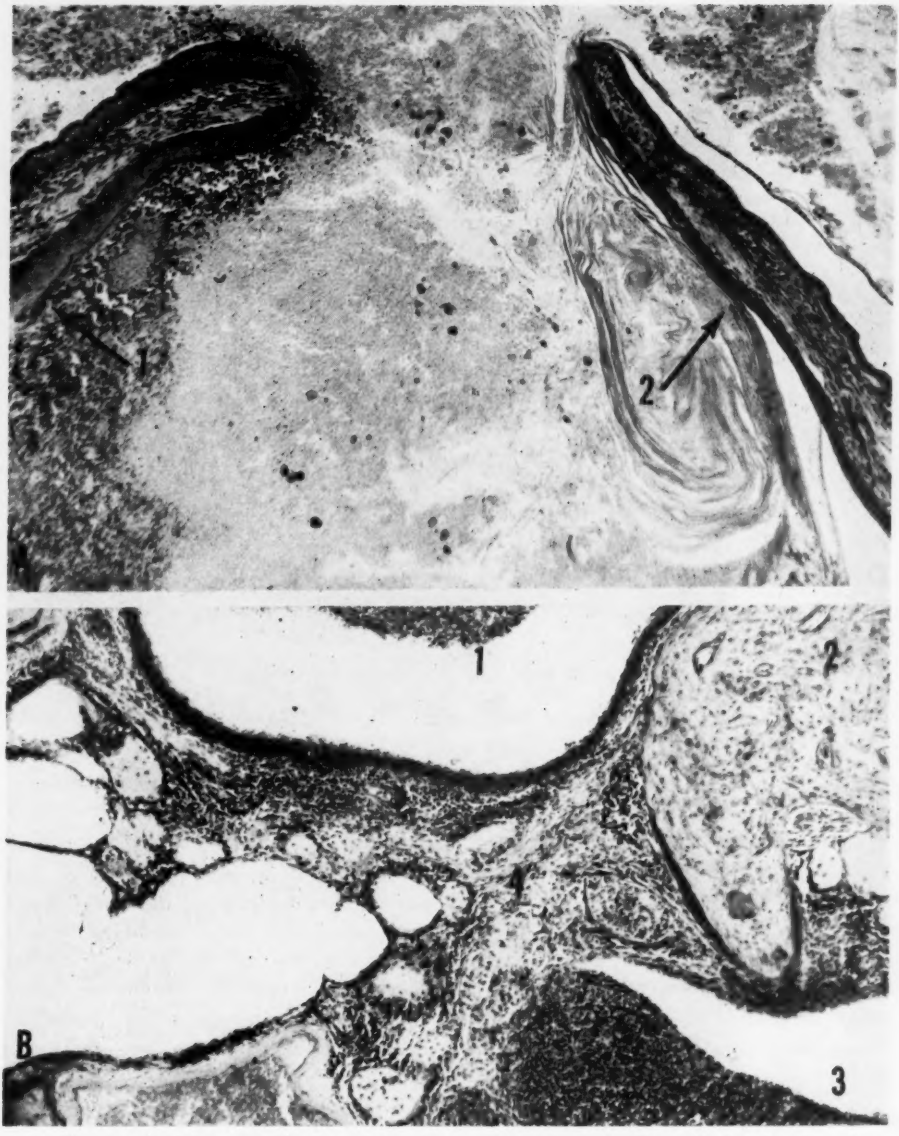


Fig. 4.—*A*, higher magnification of the abscess stoma shown in Figure 3. Immigration of the skin and epidermis of Shrapnell's membrane is evident. Pus and desquamated epithelium are demonstrated. Arrows 1 and 2 point to regions which are shown, at higher magnification, in Figures 5 and 6 respectively. Hematoxylin-eosin stain; × 120.

B, region showing the relationship of the abscess in the attic and the tympanic cavity. Hyperplastic inflammatory tissue tends to separate the two spaces. (1) Abscess. (2) Incus. (3) Pus in the tympanic cavity. (4) Hyperplastic inflammatory tissue.

pathologic studies. Several explanations have been proposed for this ingrowth of the epidermis.

There has been general agreement that an inflammatory process is the primary cause. The theory of Bezold [20] and Witt-

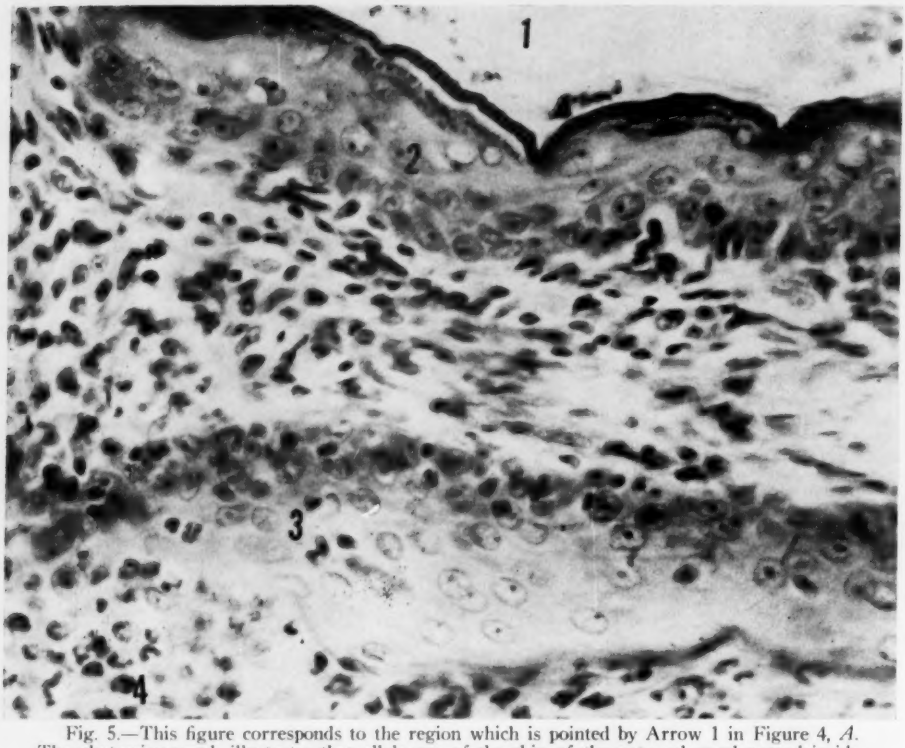


Fig. 5.—This figure corresponds to the region which is pointed by Arrow 1 in Figure 4, *A*. The photomicrograph illustrates that all layers of the skin of the external canal extend inside the abscess. (1) External canal. (2) Skin. (3) Lining of the cavity. (4) Pus and debris. Hematoxylin-eosin; × 675.

maack [14] claimed that walling off of the attic occurs and that resulting negative pressure is the essential factor in the retraction of Shrapnell's membrane, predisposing to retention of debris. That negative air pressure is an essential factor is no longer accepted.

The theory of Ojala and Saxén [16] emphasizes the importance of the corium. They reason that the formation of openings in the corium by the inflammatory process permits the basal-cell layer to proliferate through into the attic.

Rüedi [17] emphasizes the "inborn energy of growth in the epidermis of the auditory canal adjoining the tympanic membrane superiorly."

These latter two theories offer an explanation for the occasional case of attic cholesteatoma without perforation.

In our opinion there is considerable evidence [21] to show that epidermis in other parts of the body bears the same capacity for proliferation under conditions of stimulation as that of the external auditory canal and Shrapnell's membrane.

Our clinical observation has afforded evidence that the primary or predisposing factor in one type of attic cholesteatoma is the contraction of fibrous tissue and eventual retraction of Shrapnell's membrane caused by repeated inflammatory episodes. The upper posterior quadrant of the pars tensa may also be a point of origin. In such cases the inflammatory process has caused a loss of continuity of the middle fibrous layer, resulting in flaccidity which allows retraction of this quadrant towards the aditus. The precipitating factor is an acute inflammatory episode with swelling of

Fig. 6.—This figure corresponds to the region which is pointed by Arrow 2 in Figure 4, *A*. The photomicrograph illustrates that the epidermis of Shrapnell's membrane has extended into the cavity. (1) External canal. (2) Epidermis of Shrapnell's membrane. (3) Lining of the cavity. (4) Desquamated layers and debris in the cavity. Hematoxylin-eosin stain; × 825.

the epidermis, exudation, exfoliation, and retention. The retention of this debris is the factor which maintains the irritation and proliferation of the basal-cell layer. If retention is relieved, proliferation stops.

Another type of attic cholesteatoma is that produced by immigration of epidermis through a spontaneous perforation in the region of Shrapnell's membrane. The cases presented in this report clearly demonstrate the immigration of the epidermis of Shrapnell's membrane through such a perforation as an attempted reparative process. The findings demonstrate that the immigration of epidermis through a perforation is a fact, at least in experimental animals. Under conditions of chronicity and insufficient drainage a cholesteatoma would develop. These cases provide a possible explanation for the development of attic cholesteatoma after a single rupture of Shrapnell's membrane.

So far, we believe that in most of the cases the cholesteatoma develops from the epidermis, by immigration through a perforation or in cases of repeated inflammation by ingrowth of the basal cell layer secondary to an acute inflammatory episode and retention of debris in a retracted pocket.

Summary

The present status of the origin of attic cholesteatoma is briefly reviewed. The evidence points to multiple sources. Primary cholesteatoma is rare. The theory of metaplasia is not substantiated. Ingrowth of epidermis occasionally follows trauma. The majority of cases develop as a result of repeated inflammatory episodes. Two types are recognized. One is by ingrowth of epidermis through Shrapnell's membrane or less frequently by way of the posterosuperior quadrant. A second type occasionally

develops through a perforation in Shrapnell's membrane as a result of suppuration. Evidence as to the pathogenesis of this second type is presented in a series of animal ears.

950 E. 59th St. (37).

REFERENCES

1. Habermann, J.: Zur Entstehung des Cholesteatoms des Mittelohrs, Arch. Ohren- Nasen-u. Kehlkopfh. 27:42, 1888.

2. Polvogt, L. R. M.: The Pathogenesis of Cholesteatoma, Arch. Otolaryng. 9:597-601, 1929.

3. McKenzie, D.: Pathogeny of Aural Cholesteatoma, J. Laryng. & Otol. 46:163-190, 1931.

4. Müller, J.: Von der geschichteten perlmutterglänzenden Fettgeschwulst, Cholesteatoma, in Über den feineren Bau und die Formen der krankhaften Geschwülste, Berlin, G. Reimer, 1838.

5. Diamant, M.: Cholesteatoma and Chronic Otitis, Arch. Otolaryng. 47:581-589, 1948.

6. Teed, R. W.: Cholesteatoma Verum Tympani: Its Relation to the first Epibranchial Placode, Arch. Otolaryng. 24:455-474, 1936.

7. Holmes, E. M.: A Review of 303 Cases of Cholesteatoma, Ann. Otol. Rhin. & Laryng. 47: 135-143, 1938.

8. Nager, F.: The Cholesteatoma of the Middle Ear; Its Etiology, Pathogenesis, Diagnosis and Therapy, Ann. Otol. Rhin. & Laryng. 34:1249-1258, 1925.

9. Thulin, A.: Traumatische Cholesteatomata, Acta oto-laryng. 35:575-582, 1947.

10. Nilsson, G.: Attic Cholesteatoma Following Longitudinal Fracture of the Pyramid, Acta otolaryng. 36:85-91, 1948.

11. Kelemen, G.: Colesteatoma del ático de origen traumático, Bol. Liga contra cáncer 25: 31-36, 1950.

12. Escher, F.: Traumatische Cholesteatome, Pract. oto-rhino-laryng. 16:32-40, 1954.

13. Tumarkin, A.: A Contribution to the Study of Middle-Ear Suppuration with Special Reference to the Pathogeny and Treatment of Cholesteatoma, J. Laryng. & Otol. 53:685-709, 1938.

14. Wittmaack, K.: Die entzündlichen Erkrankungsprozesse des Gehörorgans, in Henke-Lubarsch' Handbuch der speziellen pathologischen Anatomie und Histologie, edited by F. Henke and O. Lubarsch, Vol. 12, Gehorörgan, Berlin, Springer-Verlag 1926.

15. Day, K. M.: Primary Pseudocholesteatoma of the Ear, Arch. Otolaryng. 34:1144-1151, 1941.

16. Ojala, L., and Saxén, A.: Pathogenesis of Middle-Ear Cholesteatoma Arising from Shrapnell's Membrane (Attic Cholesteatoma), Acta oto-laryng., Supp. 100, pp. 33-54, 1952.

17. Rüedi, L.: Pathogenesis and Treatment of Cholesteatoma in Chronic Suppuration of the Temporal Bone, Ann. Otol. Rhin. & Laryng. 66:283-305, 1957.

18. Moskowitz, M. S.; Moskowitz, A. A.; Bradford, W. L., Jr., and Wissler, R. W.: Changes in Serum Lipids and Coronary Arteries of the Rat in Response to Estrogens, A. M. A. Arch. Path. 61:245-263, 1956.

19. Schuknecht, H. F.: Techniques for Study of Cochlear Function and Pathology in Experimental Animals, A. M. A. Arch. Otolaryng. 58: 377-397, 1953.

20. Bezold, F., cited by Ojala and Saxén.[16]

21. Anderson, W. A. D., Editor: Pathology, St. Louis, The C. V. Mosby Company, 1948.

The Value of Self-Inflation of the Middle Ear

AUBREY G. RAWLINS, M.D., San Francisco

The Eustachian tubes, leading from the middle ears to the nasopharynx, are normally closed and only open with movements such as swallowing and yawning. If there is some obstruction and normal opening movements do not then open the tubes, a vacuum is produced in the middle-ear cavities and this results in impaired hearing.

There are numerous common causes of obstruction in the Eustachian tubes. Among them are hypertrophied adenoid tissue, upper respiratory infections, respiratory allergy, tumors, et cetera. If these obstructions are eliminated and not allowed to continued for a long period of time, normal physiology is restored and no permanent pathology will result.

However, there are many cases in which the cause of blockage cannot be found or where it cannot be completely eliminated. If such conditions are not properly treated, a chronic obstruction, or partial obstruction, will produce irreversible middle-ear changes with resulting impairment of hearing.

Almost since the inception of our specialty inflation of the middle ear cavities by the otologist has been practiced. In many instances, this has been, and still is, a very worth-while office procedure. However, important occasions arise, such as in air travel, where the otologist is not available to inflate the Eustachian tubes. Also, in the above-mentioned chronic cases, occasional inflation by the doctor is not sufficient to preserve hearing because the vacuum reforms fairly soon after inflation. In order to prevent irreversible middle-ear pathology in the latter situation, the Eustachian tubes must be inflated numerous times daily. It is economically unsound for the patient to be required to go to the doctor's office frequently during the 24 hours. Also, it takes too much time from both the patient and the otologist. Therefore, it is necessary, in many cases, that the patients be taught to inflate their own Eustachian tubes.

There are two ways by which the patient can accomplish this. One is the old Valsalva maneuver. In this method, the patient holds the nose tightly and makes pressure in the nasopharynx by blowing outward very strongly. This method is very useful except in conditions where the tubes are unusually tight. In such cases, the second method should be used. This maneuver is by the use of some type of rubber bag (Politzer bag) which has a tip or an adopter that fits snugly into one nostril. The tip or adapter is put in one nostril and pushed upward so that it is air-tight. The other nostril is tightly closed with the finger. The patient swallows and, at the instant of the swallow, the bag is forcibly squeezed, thus forcing air through the tubes. Swallowing a little water makes the maneuver more effective. A No. 3 ear syringe, in which a valve has been inserted, is a small and useful instrument for this purpose.

It is not always easy for the patient to learn self-inflation. It is necessary for the patient to practice in the office while the otologist observes the eardrum with a magnifying otoscope. By doing this, one can determine whether or not inflation by the patient is successful. If not, further instruction is necessary. Even fairly young children can be taught self-inflation.

Specific conditions in which self-inflation should be done are as follows:

Submitted for publication Aug. 8, 1958.

From the Department of Otolaryngology, Stanford University School of Medicine.

Read before the Section on Laryngology, Otology, and Rhinology at the 107th Annual Meeting of the American Medical Association, San Francisco, June 27, 1958.

1. For the prevention of the well-known condition of aerotitis media. This syndrome occurs in persons with improperly functioning Eustachian tubes and particularly in patients flying with a cold. By taking antihistaminics, shrinking up the nasal passages with shrinking spray, and self-inflation, aerotitis media can and should be prevented. The inflation should be done on descent as soon as the patient has the first symptoms of pain or blockage. If postponed, the valvular action of the tubes makes inflation very difficult, and also vacuum damage may have already taken place.

2. During the subacute stage of a cold where the Eustachian tubes are swollen and where the patient has a feeling of stuffiness in the ears. If before self-inflation the nose is shrunk with a spray or drops, and all the mucus drawn back and hacked up, there is very little danger of blowing infection into the middle ear. This has been shown by the experience of Army fliers. If self-inflation is practiced in this condition, many attacks of secretory otitis media will be prevented. Naturally, patients should consult with their otologist for diagnosis and instructions before this treatment is started.

3. In chronic secretory otitis media. In October, 1953, at the Academy meeting in Chicago, I reported that frequent self-inflation was an excellent addition to the treatment for this condition. After considerable more experience, I feel even more strongly that this procedure is one of the most important parts of the treatment for chronic secretory otitis media. After the office procedure of inflation, followed by paracentesis, reinflation, and spot or mass suction, the hearing usually clears up temporarily. However, since in this condition the Eustachian tubes do not open properly, the fluid keeps reaccumulating. This often goes on indefinitely. With frequent self-inflation, this reaccumulation can be prevented and hearing will return to normal. In my opinion, this is of tremendous importance.

4. In chronic stuffy ears without fluid. In this group, we have patients whose tubes function improperly but not badly enough to make fluid collect in the middle ear. There is a feeling of stuffiness in the ears a large part of the time and there is a gradual development of middle-ear type of impaired hearing. Some of the patients have found out, by their own experience, that holding the nose and blowing will give them some relief. The tubes are not as tight as in the secretory cases, and the Valsalva maneuver usually suffices in this group. Being in this category myself, I can say from first-hand experience that self-inflation in this condition is very effective. As was previously stated, the patient should consult with his otologist for diagnosis and instruction before any self-treatment is carried out.

Summary and Conclusions

The value of self-inflation of the Eustachian tubes has been considered. Specific circumstances are given where this procedure is indicated. It is my opinion that many cases of middle-ear type of impaired hearing could be prevented if more patients, in the categories discussed, would frequently hold their noses and blow or would carry a small rubber bag and consistently inflate their middle-ear spaces.

384 Post St. (8).

The Localization of Acetylcholinesterase in the Cochlea

HAROLD F. SCHUKNECHT, M.D.; JOHN A. CHURCHILL, M.D., and ROSEMARY DORAN, B.S., Detroit

Introduction

Recently we reported the results of experiments[3,17] demonstrating that acetylcholinesterase (AChE) was present in the nerve fibers within the cochlea. Koelle's method for the histochemical detection of AChE was employed. The technique was applied to the isolated cochleas of cats after removal of the outer bony wall. Portions of the osseous spiral lamina and organ of Corti were then teased onto glass slides for study. In brief, the histochemical method, described fully elsewhere,[8-10] consisted of incubating tissues in a solution of acetylthiocholine in the presence of cupric ion. The thiocholine liberated by enzymatic hydrolysis immediately formed a precipitate with cupric ions. The product, copper thiocholine, was converted later to copper sulfide when the specimen was treated with ammonium sulfide. Thus, copper sulfide, a stable and visible precipitate, was seen where AChE had been located. We satisfied ourselves that AChE was the material present in the cochlea by treating specimens with diisopropyl fluorophosphate (DFP), which inactivated the nonspecific cholinesterases, and by incubating other specimens with butylthiocholine iodide which favored the detection of nonspecific cholinesterases. A dense black stain, indicating AChE activity, was found on the nerve fibers located within and near the organ of Corti. In some regions, particularly at the nerve endings, the stain was as dense as that seen on muscle end-plates. We were able to confirm the previous reports by Martini and by Gisselsson that cholinesterase was present in the cochlea. Martini[11,12] demonstrated that acetylcholine was present in the inner ear perilymph, and later Gisselsson[6] detected cholinesterase in both perilymph and endolymph of the cochlea by incubating measured volumes of these fluids with acetylcholine of known concentration and comparing the contraction of dorsal muscle of the leech and frog rectus muscle immersed in the solution. The magnitude of the contraction of the indicator muscles in the solution yielded evidence that acetylcholinesterase was present in the cochlear fluids.

We reasoned that the dense staining from AChE activity in the cochlea might be related to the efferent nerve fibers of the olivocochlear bundle, rather than to the hair cell or the dendritic ending of the spiral ganglion cells. This reasoning was based upon reports of others indicating that AChE activity was very weak in other types of afferent nerve fibers. The olivocochlear bundle of Rasmussen is a tract originating from cell bodies near the superior olivary bodies which passes into the contralateral cochlea. The ganglion cell bodies of the olivocochlear bundle lie in the medulla, medial to the superior olivary nucleus and dorsal to the nucleus of the trapezoid body.[14,15] The tract decussates in the floor of the fourth ventricle near the colliculi of the facial nerves and passes into the inferior division of the vestibular nerve. Passing through the saccular ganglion, the bundle divides into three fascicles which, upon entering the spiral ganglion canal, are known as the intraganglionic fascicles. The fascicles course along the spiral ganglion from which fibers are directed toward the organ of Corti. The precise termination of these fibers has not been determined.

Submitted for publication Dec. 17, 1958.

From the Divisions of Otolaryngology and Neurology, Henry Ford Hospital.

This work was supported by a Research Grant B-734-C3 from the National Institute of Neurological Diseases and Blindness of the U. S. Public Health Service.

Purpose of the Study

The present experiments were designed to find the precise localization of AChE within the cochlea and to determine whether the presence of AChE in the cochlea was dependent upon the efferent, olivocochlear bundle.

The Sets of Experiments

Experiments for the detection of AChE on the nerve fibers in the cochlea were made after (1) section of the acoustic nerve, both afferent and efferent fibers being cut; (2) section of the olivocochlear tract, efferent fibers only being sectioned, and (3) destruction of the hair cells. The three sets of experiments will be considered separately after a description of the methods used and results obtained from examination of the control ears. In each cat the lesion was created in the right ear with the left serving as a control. The control ears provided information regarding the normal distribution of AChE in the cochlea. All operations were performed using aseptic techniques and intraperitoneal pentobarbital (Nembutal) anesthesia. The survival time of each animal was varied to suit the experimental needs. One to five weeks after operation animals were decapitated and the inner ears removed immediately from the skull. The stapes was removed from the oval window, and the round window membrane incised. A short length of polyethylene tubing * was sealed into the round window niche with dental cement. The cochleas were perfused through the tubing with the various solutions, care being taken to avoid injection of air bubbles. Injections were made slowly to avoid rupture of Reissner's membrane or avulsion of the organ of Corti. Thus, the solutions traveled through the scala tympani, the helicotrema, the scala vestibuli, to emerge from the oval window. Both cochleas and also a section of skeletal muscle were treated to demonstrate AChE activity. The muscle sections, showing a dense stain, in motor end-plates, served as proof that proper chemical reactions had occurred. A separate muscle section was treated similarly, except that it was not exposed to the substrate acetylthiocholine. These sections served as controls against falsely positive reactions; and in no instance was there any precipitate in these control sections. After the cochleas were perfused with the reagents, they were perfused with 10% formalin, decalcified, imbedded in celloidin, sectioned serially at a thickness of 20μ, mounted on glass slides, and counterstained with hematoxylin and eosin.

* Clay Adams PE 50, inside diameter 0.023 in., outside diameter 0.038 in.

Results of Perfusions in Control Ears

A greater concentration of copper sulfide stain was seen on fibers located near the inner hair cells than on the outer hair cells (Fig. 1*A*). This difference in staining may be related to the fact that there is a greater number of nerve fibers in the inner spiral bundle than in the outer. The stain was seen only on fibers near the inner hair cell row in some cochleas. The stain was densest in the upper basal turn, decreasing progressively toward the basal and apical ends. Stain was present on fibers crossing the midportion of Corti's tunnel and those on the floor of the tunnel. Nerve endings were also densely stained; in many areas they were stained as richly as were muscle end-plates. One could not determine whether the precipitate was on the surface or within the nerve fiber endings, for the tissues were too thick (20μ). Stain usually was found on the endings of the fibers and along the fibers as far as one-third to one-half their length toward the spiral ganglion. The spiral ganglion cells and nerve fibers in the ganglion canal were not stained. One could not conclude that these structures were free of acetylcholinesterase, for the solutions probably did not penetrate to the ganglion. An extremely interesting finding was the presence of stain within small canals in the bony inferior shelf of the osseous spiral lamina. These canals appeared to connect the scala tympani with spaces about the nerve fibers for the flow of perilymph or

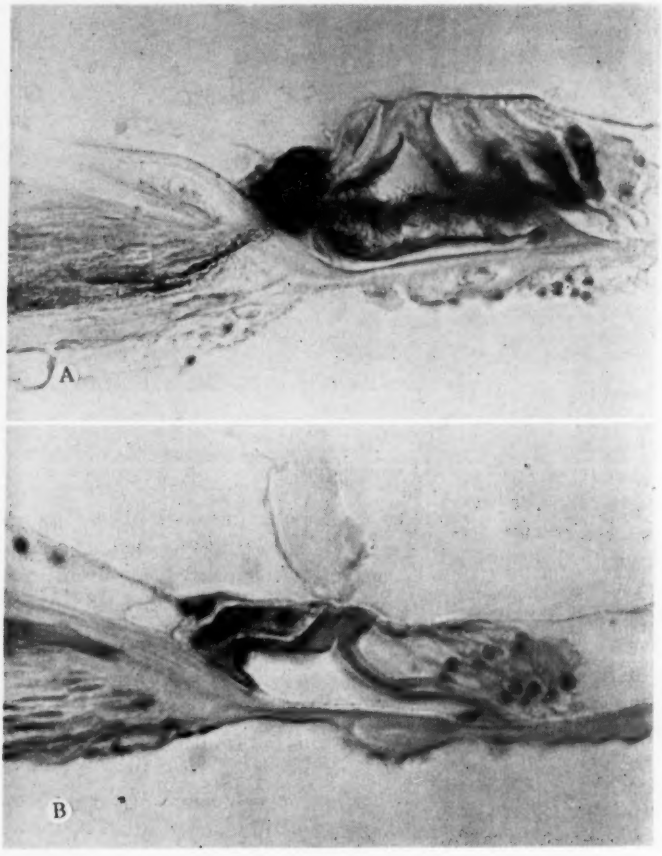


Fig. 1 (Cat 6).—This animal was killed 30 days after section of the right acoustic nerve. *A*, stain on nerve fibers in organ of Cotri of left (control) ear. *B*, small deposit of stain in area of inner spiral bundle of right (experimental) ear. Normal hair cells.

endolymph. Stain located between the outer Deiter's cells presumably was located on nerve fibers entering the outer spiral bundles. The stain was not seen in the region of the inner or outer sulcus cells, Hensen's cells, or stria vascularis. Occasionally stain was located on the pillar cells, particularly the surface of the pillars bordering the tunnel; but this occurred only when there was also a dense stain on nerve fibers within the tunnel. Needle-shaped crystals occasionally seen on the nerve fibers were thought to be copper thiocholine which had escaped complete conversion to copper sulfide.[20]

Section of the Acoustic Nerve

A posterior fossa craniotomy was performed in six cats, and the right acoustic nerves were exposed and crushed against the inferior rim of the internal auditory meatus with a blunt instrument. In this procedure efforts were made to spare the cochlear artery from damage. In four animals, allowed to survive 14 days, the spiral ganglia of the right cochleas were found to be in late stages of degeneration (Table 1), the nuclei of the ganglion cells showing pyknotic, karyorrhectic, or karyolytic changes. Nuclear debris from destroyed ganglion cells was scattered about. Spiral ganglion cell degeneration was complete in the two animals allowed to live 23 and 30 days after operation. The condition of the efferent nerves in the experimental and control cochleas was evaluated by comparing the hematoxylin and eosin staining characteristics of the nerve fascicles at the

TABLE 1.—*Acoustic Nerve Section: Summary of Results of Sectioning Right Acoustic Nerve in Six Cats**

Cat No.	Survival Time, Days	Degen. Afferents	Degen. Efferents	Decrease in Cochlear Stain for AChE
1	14	Moderate	Great	Slight
2	14	Moderate	Great	Moderate
3	14	Moderate	Great	Moderate
4	14	Moderate	Great	Great
5	23	Great	Great	Great
6	30	Great	Great	Great

* The left ears appeared normal and showed normal AChE activity. The afferent and efferent nerves were degenerated in all the right ears and in these the AChE stain was decreased.

point where they leave the saccular ganglion in the internal auditory meatus. In all six animals the efferent nerves on the right were less deeply stained than those on the control, left side, indicating degenerative changes in the right ears. The organs of Corti were morphologically normal in these six animals.

The amount of copper sulfide stain was decreased in the ears operated on and normal in all control ears. In the denervated cochleas there was very little stain on the nerve fibers or on the nerve endings, and that which remained was condensed into compact punctate accumulations (Fig. 1*B*). The least stain was found in the ears operated on in the two animals surviving 23 and 30 days respectively. This experiment showed that there was a great decrease in AChE activity in ears with degeneration of both the afferent and efferent nerve tracts. The experiment, however, did not produce information from which one could tell whether the loss of AChE was due to degeneration of the afferent or of the efferent fibers.

Other experiments have shown that section of a motor nerve results in a decrease of ACh or AChE in the degenerating nerve itself, but that the concentration of these enzymes in the muscle end-plates diminishes little or not at all over a period of many months.[1,4,13,16]

Section of the Olivocochlear Bundle

The olivocochlear bundle of the right ear was cut by an incision made in the right

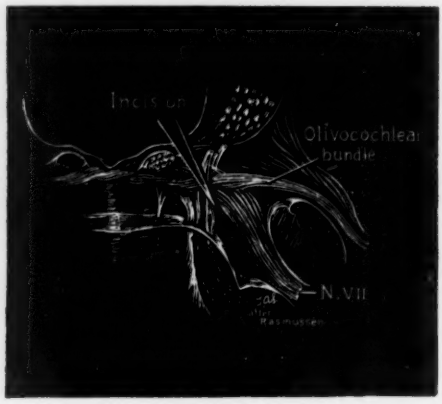


Fig. 2.—Diagrammatic cross section of medulla showing site and angle of incision. Facial spasms occurring at the time of operation was an indication of a properly located incision.

sulcus limitans in the floor of the fourth ventricle, 10 mm. from the obex (Fig. 2). The animals were allowed to survive for periods of time varying from 7 to 35 days, after which they were decapitated. When the brains were removed the acoustic nerves were cut at the internal auditory meatuses so that part of the trunk of each nerve was left intact with the temporal bone specimen. The brains were fixed in 10% formalin, and then frozen sections of the brain stem were cut. The sections were stained with Sudan Black B and mounted in Apathy's syrup.

The size of the lesion in both the vertical and lateral planes varied considerably in the different animals. Only a narrow zone of gliosis in the path of the knife cut was seen in some specimens, while the area of degeneration extended a millimeter or more from the incision in others. Degeneration of the efferent fibers was seen in brain sections in 9 of 11 cats, but in 3 of these 9 the degeneration extended no further than the saccular ganglion. Degenerative changes in the olivocochlear bundle were observed most readily in the internal auditory meatus where the bundle leaves the saccular ganglion to enter the modiolus, the characteristic change being a decrease in affinity for hematoxylin and eosin (Compare Figs. 3 and 4).

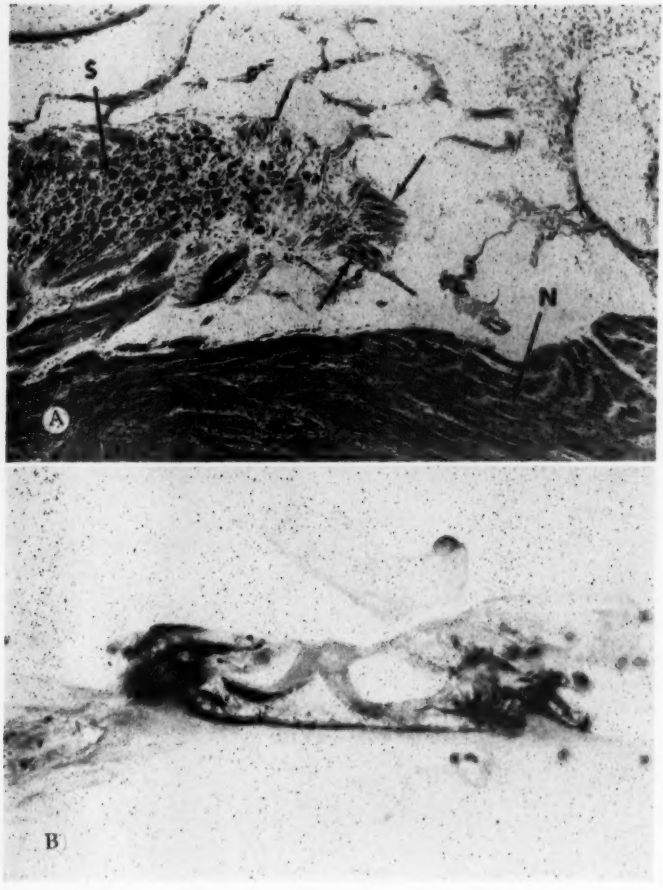


Fig. 3 (Cat 8).—Photomicrographs from normal left ear of Cat 8. *A*, view in internal auditory meatus showing saccular ganglion (*S*), cochlear nerve (*N*) and olivocochlear bundle (between two arrows). *B*, AChE stain in organ of Corti is normal.

Acetylcholinesterase was markedly decreased in six cochleas in which the efferent fibers had degenerated (Figs. 5 and 6). The efferent fibers had remained intact in four ears in which AChE in the organ of Corti persisted (Table 2). In one ear (Cat 14) the nerve was inadvertently avulsed from the internal auditory meatus and so was not available for study.

In 4 of the 11 brains the lesions were extensive enough to involve some of the olivocochlear bundle to the opposite (left) ear. However, in these animals the olivocochlear bundle appeared to be normal at the level of the saccular ganglion.

The stain representing AChE activity was normal in amount in all of the control (left) ears. There appeared to be no reason why an apparently successful section of the efferent bundle in the medulla did not result in degeneration of the bundle at the level of the saccular ganglion in three cats. Survival time following section of the bundle did not appear to be a factor. Cat 7, allowed to survive only seven days, already had degeneration of the efferent bundle in the internal auditory meatus and decreased staining in the cochlea.

Loss of Hair Cells

One of us (H. F. Schuknecht,[18]) has found that a single injection of streptomycin sulfate (0.5 gm. per cubic centimeter) into the auditory bulla of the cat resulted in

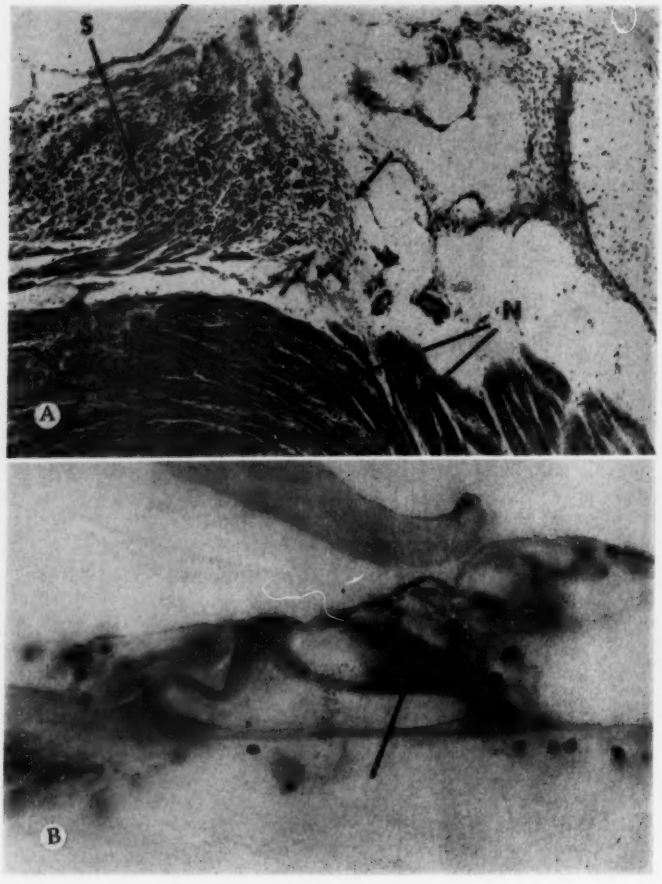


Fig. 4 (Cat 8).—Photomicrographs from the right (experimental) ear of Cat 8 killed 14 days after cutting right olivocochlear bundle. *A*, view in internal auditory meatus showing saccular ganglion (*S*), cochlear nerve (*N*) and degenerated olivocochlear bundle (between two arrows). *B*, only a small discrete deposit of stain (arrow) indicating AChE is present in region of first outer spiral bundle. (For details of criteria used to establish comparative stain densities, see Legend, Fig. 7.)

degeneration of hair cells of the organ of Corti. The typical changes observed were loss of hair cells and only minor alterations of the supporting cells and nerve endings. In several other experiments we showed that the spiral ganglion cell bodies remained intact as long as the supporting elements of the organ of Corti were not damaged, even though the hair cells were totally lost. We did not determine whether or not the nerve endings were always intact in such ears.[19]

Streptomycin injections were made into the right tympanic bullae of four cats. The animals were kept 14 to 21 days and then decapitated (Table 3). The cochleas removed from the skull were treated for the detection of AChE. The severity of injury to the organ of Corti varied considerably in different ears and in different regions of the same ear. In all four ears there was some damage to supporting cells of the organ of Corti and presumably to the dendritic endings of afferent nerve fibers. In one ear the organ of Corti was severely atrophied and the spiral ganglion was partially degenerated. As would be expected, there was a great decrease in stain in this ear, as well as an over-all decrease in the amount of stain in the other three ears. In each ear, however, there were areas of atrophy of the organ of Corti with loss of hair cells where AChE remained (Fig. 7). Thus, AChE did not appear to depend upon the integrity of the hair cells.

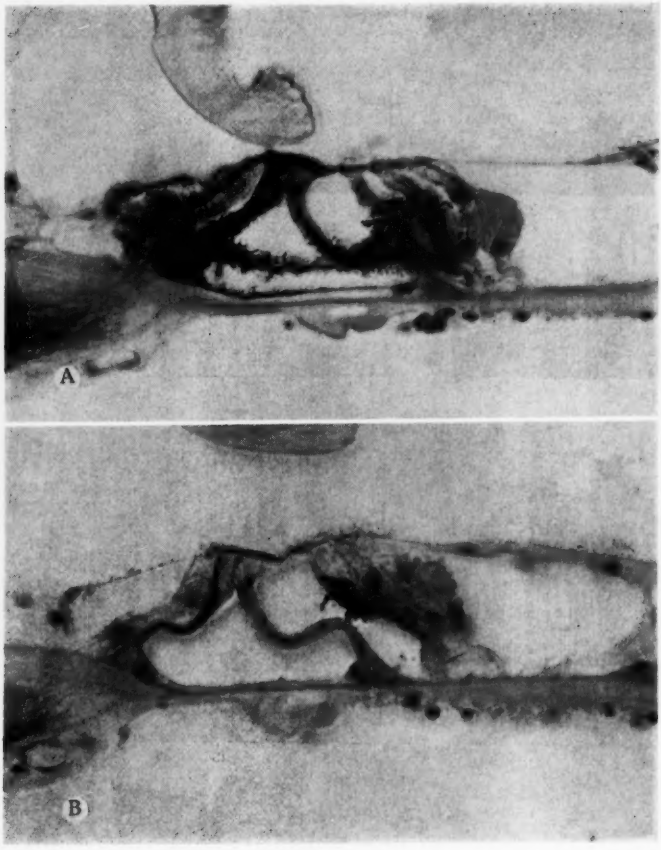


Fig. 5 (Cat 16).—This cat was killed 28 days after the right olivocochlear bundle was cut. The stain for AChE is normal in the left (control) organ of Corti shown above, and greatly decreased in the right (experimental) organ of Corti shown below.

Comment

The abundance of acetylcholinesterase found at the bases of hair cells in the organ of Corti has led us to assume that acetylcholine is important in the transmission of impulses at hair cell-acoustic nerve junctions. This assumption is justified by the evidence regarding the function of acetylcholine in the transmission of nerve impulses elsewhere in the nervous system. Others [2,7] have found that concentrations of acetylcholinesterase parallel those of acetylcholine in various locations.

Since acetylcholinesterase was found markedly diminished in cochleas after successful section of their olivocochlear tracts in the medulla but remained after degeneration of hair cells, we concluded that acetylcholinesterase in the cochlea depended upon the integrity of the olivocochlear bundle. One might expect, a priori, that the acetylcholinesterase found in the cochlea would be related to efferent olivocochlear fibers, since afferent neurons generally have been found to contain only low concentrations of acetylcholine. The olivocochlear bundle appears to be a feed-back pathway originating from a relay point (the retro-olivary nucleus of the superior olivary body) in the contralateral side of the medulla and ending upon the auditory hair cells in the organ of Corti. There is reason to believe that impulses transmitted by this tract modulate the response levels of hair cells or endings of the acoustic nerves, for Galambos [3] has observed inhibition of sound-induced action

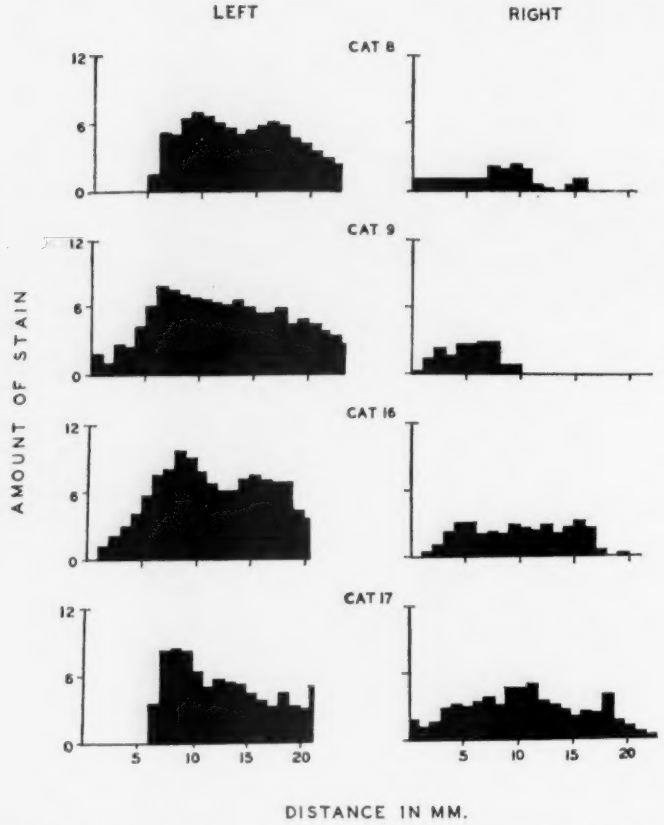


Fig. 6.—Charts showing relative amounts of stain in the cochleas of four cats in which the olivocochlear bundle was cut on the right side. The cochleas were graphically reconstructed and an estimate was made of the density of stain throughout the length of the cochlea. The stain was rated as being absent, light or dense, with values of 0, 1, and 2 respectively, for each of six structures and a total value given for the cochlear duct at each point where it was sectioned. Below is shown a rating of 1 in the 15 mm. region of the right ear of Cat 8. (See photomicrograph, Fig. 4.)

Location of Nerve Fibers	*Density of Stain*
Osseous spiral lamina	0
Inner spiral bundle	0
Tunnel of Corti	0
First outer spiral bundle	1
Second outer spiral bundle	0
Third outer spiral bundle	0
Total rating	1

On the ordinate is the stain density rating and on the abscissa the distance in millimeters as measured along the organ of Corti. In the normal left ear the concentration is greatest in the upper basal turn in about the 7 mm. to 11 mm. regions. The amount of stain diminishes rapidly from this region in a basalward direction, and in some ears is totally absent in the hook end of the cochlea. Although the stain diminishes progressively toward the apex, considerably more exists at the apex than at the base. The amount of stain is considerably reduced in the right ears which were de-efferentized.

potentials in the acoustic nerve when the olivocochlear tract was stimulated. One can reason that impulses transmitted to the endings or paraterminal boutons of the olivocochlear fibers permit liberation of acetylcholine. The released acetylcholine then

TABLE 2.—*Section of Efferent Bundle: Results of Cutting the Olivocochlear Bundle on the Right Side in Eleven Cats* *

Cat No.	Survival Time, Days	Degen. Efferents	Decrease in Cochlear Stain for AChE
7	7	Great	Moderate
8	14	Great	Great
9	14	Great	Great
10	18	None	None
11	18	None	None
12	20	Great	Moderate
13	20	None	None
14	21	(Artefact)	Great
15	21	None	None
16	28	Great	Great
17	35	Great	Great

* In the six ears in which the olivocochlear bundle was degenerated, there was also a decrease in cochlear stain for AChE. The four ears with a normal appearing olivocochlear bundle had normal stain density. The nerve was inadvertently avulsed from the internal auditory meatus of Cat 14 and was not available for study.

TABLE 3.—*Intratympanic Streptomycin: Summary of Findings in Four Ears in Which Hair Cells Were Damaged* *

Cat No.	Survival Time, Days	Degen. Spiral Ganglion	Damage to Supporting Cells	Absence of Hair Cells	Decrease in Stain for AChE
18	20	None	Basal Turn	Basal Turn	None
19	24	None	Basal Turn	All Turns	Moderate
20	14	None	All Turns	All Turns	Moderate
21	24	Moderate	All Turns (Severe)	All Turns	Great

* In all ears there were regions where the hair cells were missing and AChE normal. There was damage also to nerve fibers in Cats 19, 20, and 21, which accounts for the generalized decrease in AChE in these ears.

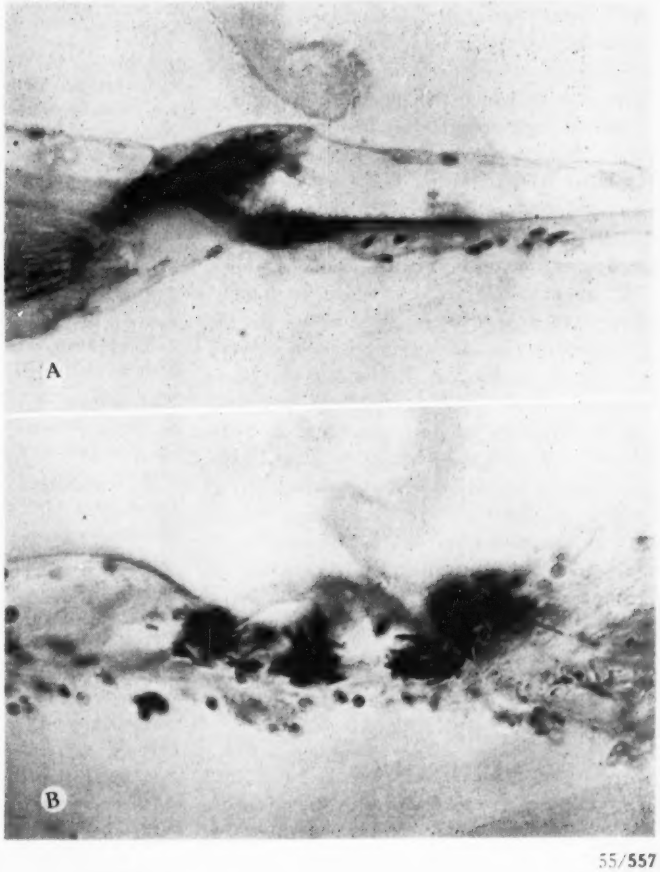


Fig. 7.—AChE remaining after loss of hair cells. *A* (Cat 18), killed 20 days after injection of streptomycin sulfate into auditory bulla. There is severe atrophy of the organ of Corti, but a dense concentration of stain exists in the region of the inner spiral bundle and on the basilar membrane. *B* (Cat 20), severe atrophy of organ of Corti but dense stain located in region of spiral bundles and tunnel fibers.

alters the state of excitation of the endings of the acoustic nerves or the hair cells before it is hydrolyzed by acetylcholinesterase.

Summary and Conclusions

We have previously published the results of studies showing that acetylcholinesterase is present along nerve fibers and in dense accumulations at the bases of the hair cells in the organ of Corti of the cat. In the present study fresh cat cochleas were perfused with acetylthiocholine in the presence of cupric ions, the solutions passing from the round window through the helicotrema and out the oval window. The cochleas were fixed, decalcified, sectioned, and counterstained. The copper sulfide deposits resulting from activity of acetylcholinesterase were found to be markedly decreased in six cochleas in which the olivocochlear bundle had been successfully sectioned in the medulla. Acetylcholinesterase was not decreased in any of the control ears or in the two ears in which the olivocochlear bundle had not degenerated after unsuccessful section. The esterase remained in the neighborhood of hair cells that had been destroyed by streptomycin in three cats. It was concluded that the presence of acetylcholinesterase in the cochlea was dependent upon the integrity of the olivocochlear bundle. The evidence revealed that fibers of the olivocochlear bundle terminate upon or very close to the cochlear hair cells, and that these fibers are cholinergic. Acetylcholine liberated at the endings of the olivocochlear tract fibers may alter the excitability state of the hair cells or the acoustic nerve endings.

The amacrine cells of the retina, which are known to contain acetylcholinesterase,[21] may subserve a function similar to that of Rasmussen's bundle in the cochlea. Perhaps other sensory end-organs should be investigated for cholinergic efferent nerve fibers.

Dr. Joyce White and Dr. Donald Elliott assisted with many phases of the experimentation.

Henry Ford Hospital (2).

REFERENCES

1. Brooks, V. B., and Myers, D. K.: Cholinesterase Content of Normal and Denervated Skeletal Muscle in the Guinea Pig, J. Physiol. 116:158-167, 1952.

2. Burgen, A. S. V., and Chipman, L. M.: Cholinesterase and Succinic Dehydrogenase in the Central Nervous System of the Dog, J. Physiol. 114:296-305, 1951.

3. Churchill, J. A.; Schuknecht, H. F., and Doran, R.: Acetylcholinesterase Activity in the Cochlea, Laryngoscope 66:1-15, 1956.

4. Couteaux, R., and Nachmansohn, D.: Changes of Choline Esterase at End Plates of Voluntary Muscle Following Section of Sciatic Nerve, Proc. Soc. Exper. Biol. & Med. 43:177-181, 1940.

5. Galambos, R.: Suppression of Auditory Nerve Activity by Stimulation of Efferent Fibers to Cochlea, J. Neurophysiol. 19:424-437, 1956.

6. Gisselsson, L.: Experimental Investigation into the Problem of Humoral Transmission in the Cochlea, Acta oto-laryng., Supp. 82, pp. 1-78, 1950.

7. Hellauer, H.: Über den Cholinesterasegehalt nervösen Gewebes, Arch. f. ges. Physiol. 242:382-388, 1939.

8. Koelle, G. B., and Friedenwald, J. S.: A Histochemical Method for Localizing Cholinesterase Activity, Proc. Soc. Exper. Biol. & Med. 70:617-622, 1949.

9. Koelle, G. B.: The Histochemical Differentiation of Types of Cholinesterase and Their Localizations in Tissues of the Cat, J. Pharmacol. & Exper. Therap. 100:158-179, 1950.

10. Koelle, G. B.: The Elimination of Enzymatic Diffusion Artifacts in the Histochemical Localization of Cholinesterases and a Survey of Their Cellular Distributions, J. Pharmacol. & Exper. Therap. 103:153-171, 1951.

11. Martini, V.: Presenza di colinesterasi nella perilimfa dell'orecchio interno del piccione, Boll. Soc. ital. biol. sper. 16:70-72, 1941.

12. Martini, V.: Liberazione di sostanza acetilcolino-simile nell'orecchio interno durante la stimolazione sonora, Arch. sc. biol. 27:94-100, 1941.

13. Nachmansohn, D., and Hoff, E. C.: Effects of Dorsal Root Section on Choline Esterase Concentration in Spinal Cord of Cats, J. Neurophysiol. 7:27-36, 1944.

14. Rasmussen, G. L.: The Olivary Peduncle and Other Fiber Projections of the Superior Olivary Complex, J. Comp. Neurol. 84:141-220, 1946.

15. Rasmussen, G. L.: Further Observations of the Efferent Cochlear Bundle, J. Comp. Neurol. 99:61-74, 1953.

16. Sawyer, C. H.: Cholinesterases in Degenerating and Regenerating Peripheral Nerves, Am. J. Physiol. 146:246-253, 1946.

17. Schuknecht, H. F.; Churchill, J. A., and Doran, R.: Acetylcholinesterase and the Olivocochlear Tract of Rasmussen, Tr. Internat. Conf. Audiology; abstracted, Laryngoscope, to be published.

18. Schuknecht, H. F.: Ablation Therapy in the Management of Ménierè's Disease, Acta otolaryng. Supp. 132, pp. 1-42, 1957.

19. Schuknecht, H. F.: Lesions of the Organ of Corti, Tr. Am. Acad. Ophth. 57:366-383, 1953.

20. Zajicek, J.; Sylven, B., and Datta, N.: Attempts to Demonstrate Acetylcholinesterase Activity in Blood and Bone-Marrow Cells by a Modified Thiocholine Technique, J. Histochem. 2:115-121, 1954.

21. Koelle, G. B.; Wolfand, L.; Friedenwald, J. S., and Allen, R. A.: Localization of Specific Cholinesterase in Ocular Tissues of the Cat, Am. J. Ophth. 35:1580-1584, 1952.

Congenital Hereditary Kidney Disease and Hearing Loss

A Case History

ROBERT E. KLOTZ, M.D., Boston

This paper is presented to acquaint the otolaryngologist with a relatively rare syndrome consisting of congenital familial kidney disease and nerve hearing loss and to report a case that was seen in the Winthrop Foundation of the Massachusetts Eye and Ear Infirmary.

In reviewing the literature it is possible to find a relatively abundant amount of material pertaining to hereditary kidney disease, but very little reporting an associated deafness. Hereditary kidney disease in any form is very unusual.[1]

Guthrie[2] in 1902 described a family in which 12 members had hematuria, either gross or microscopic. These members were reevaluated by Kendall and Hertz, Hurst, and Alport,[3] who observed that the hematuria was often intermittent. They also added some more members, seven males and eight females. An associated nerve deafness was usually found in the males, and they died of chronic renal disease before the age of 20. Only an occasional case of deafness was found in the afflicted females.

Reyersback and Butler[4] in 1954 described a family in which 22 members had nephropathy and hematuria. Deafness was found in eight males who died in early life, four living affected males, and two living affected females. Three affected males had no deafness. Females appeared to transmit the disorder.

Sturtz and Burke[5] in 1956 added another family with data of a similar nature.

The affected females apparently live through a normal life span, but tend to develop hypertension and toxemia of pregnancy.

Perkoff et al.,[1] described a family having hereditary pyelonephritis. They studied 134 members and found 44 who appeared affected and 20 who apparently carried the trait. Seven males, six of whom had nerve deafness, died between the ages of 22 and 34. Of the living members of the family, 3 "normal" females, 1 male carrier, and 10 diseased males had nerve deafness. In these cases, early symptoms included bilateral flank pain, weight loss, nocturia, and dysuria, while in previous reports the disease was usually asymptomatic and diagnosed only by routine urinalysis.

Sohar[6] in Israel investigated 10 members of a Jewish family in whom the mother of four sons was deaf and had albuminuria and casts. The four sons, aged 14 to 18, had albuminuria, hematuria, and inner-ear deafness with recruitment. Spherophakia was also noted in two of the patients. Two maternal uncles, who died in Iraq in their early 20's, had been deaf.[6]

Goldbloom et al.,[7] in 1957, reported three children with kidney disease and deafness. One infant weighed 1.4 lb. at birth. Death was due to uremia at the age of 5 years. His sibling died of uremia at the age of 7 years. He also had a hearing loss. One sibling, still living at the age of 11, had hematuria and a nerve deafness.

The renal pathology is not clearly understood. Guthrie[2] reports that at autopsy shrunken white kidneys were found. Reyersbach and Butler's[4] case showed "shrunken kidneys." A renal biopsy from one of their patients, showed blood in Henle's loops and

Submitted for publication July 29, 1958.

convoluted tubules. Goldbloom et al. found granular contracted kidneys in their first case and cystic kidneys with tubular atrophy and chronic pyelonephritis in their second case.

The case now being reported showed chronic glomerular nephritis at necropsy. Unfortunately, none of the temporal bones were studied. Sohar's observation of recruitment points a finger toward the end-organ as the site of pathology responsible for the nerve deafness. In the first case of Goldbloom it was noted that dorsal and ventral cochlear nuclei and tracts were normal. Observations have been made regarding deafness associated with nonfamilial kidney disease, and the pathology has been attributed to cochlea. Hemorrhages have been found in the labyrinth associated with nephritis.[8]

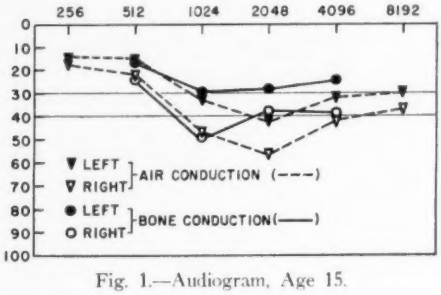


Fig. 1.—Audiogram, Age 15.

Report of Case

When first seen at the Massachusetts Eye and Ear Infirmary in March, 1954, a 15-year-old white boy complained only of hearing loss, and faint intermittent tinnitus of two and a half months' duration. He noted that yawning and coughing occasionally would improve his hearing temporarily.

Past history provided the following information:

He was born of a full-term normal pregnancy and delivery. He enjoyed an uncomplicated neonatal period. At the age of 4 years he was hospitalized for a streptococcal throat infection. A short while after discharge he had a relapse and was readmitted. His father was not told of any kidney disease at that time. He had a tonsillectomy and adenoidectomy one year later.

He has had chickenpox, whooping cough, measles, and mumps without any apparent complications or sequelae.

On physical examination, the right Eustachian tube could not be politzerized, the Weber lateralized

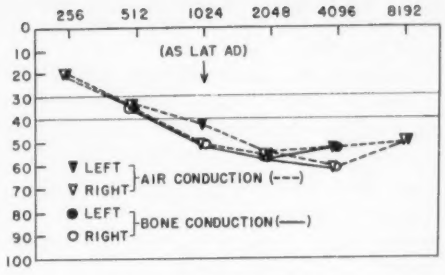


Fig. 2.—Audiogram, Age 17.

to the right side, and the Rinnes were a decreased positive. On March 19, he was again seen, and both Eustachian tubes were easily politzerized. An audiogram at this time showed a nerve type deafness (Fig. 1). Lip reading and auditory training were advised.

The patient was next seen Sept. 11, 1956, at which time he felt his hearing loss was progressing. He had stopped lip reading classes the previous year. His grades were dropping in school, and he was chronically tired. Ear, nose, and throat examination was normal except for pallor of the mucous membranes and skin, and the nerve hearing loss, which had increased (Fig. 2). Hematology done that afternoon proved an anemia with a RBC of 2,400,000, with slight variation in size, a hemaglobin of 7.8 gm., and a normal white count and differential.

The patient was transferred to the medical clinic, where a diagnosis of chronic glomerular nephritis was made. During the following four months he went progressively downhill. All urinalyses showed albuminuria and hematuria, varying from 5 to 95 RBC/hpf. In spite of many overnight stays in the hospital with multiple blood transfusions, he died in uremia Jan. 29, 1957.

Necropsy reported chronic glomerular nephritis, hemorrhagic pneumonitis, pulmonary edema, hydrothorax, gastritis, ascites, hypertensive heart disease

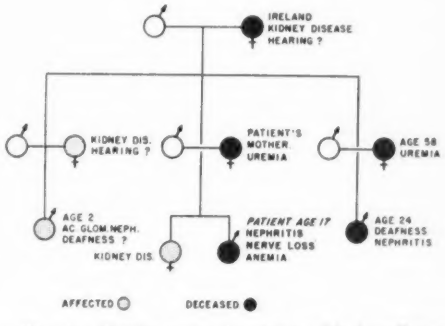


Fig. 3.—Family tree of patient with hereditary kidney disease and deafness.

with cardiomegaly, splenomegaly, and anemia. Unfortunately, the temporal bones were not obtained for study.

Studying his family history, which was difficult to obtain because of reluctance on the part of the family, we found kidney disease in eight members, three of whom were males who had hearing loss (Fig. 3). One male cousin died at the age of 24, and another, aged 3 years, has acute glomerular nephritis and is questionably hard of hearing. A sister, aged 24, has kidney disease, but no hearing loss.

Summary

The syndrome of hereditary kidney disease and nerve deafness has been reviewed, and a case reported.

243 Charles St. (14).

REFERENCES

1. Perkoff, G. T.; Stephens, F. E.; Dolowitz, D. A., and Tyler, F. H.: A Clinical Study of Hereditary Interstitial Pyelonephritis, A. M. A. Arch. Int. Med. 88:191-200, 1951.

2. Guthrie, L. G.: Idiopathic or Congenital Hereditary and Familial Hematuria, Lancet 1:1243-1246, 1902.

3. Alport, A. C.: Hereditary Familial Congenital Haemorrhagic Nephritis, Brit. M. J. 1:504-506, 1927.

4. Reyersbach, G. C., and Butler, A. M.: Congenital Hereditary Hematuria, New England J. M. 251:377-380, 1954.

5. Sturtz, G. S., and Burke, E. C.: Hereditary Hematuria, Nephropathy and Deafness, New England J. M. 254:1123-1126, 1956.

6. Sohar, E.: Renal Disease, Inner Ear Deafness, and Ocular Changes, A. M. A. Arch. Int. Med. 97:726-630, 1956.

7. Goldbloom, R. B.; Fraser, F. C.; Waugh, D.; Ceranorutch, M., and Wigglesworth, F. W.: Hereditary Renal Disease Associated with Nerve Deafness & Ocular Lesions, Pediatrics 20:241-252, 1957.

8. Politzer, A.: Text-Book of the Diseases of the Ear for Students and Practitioners, Ed. 4, translated and edited by M. J. Ballin and C. L. Heller, Philadelphia, Lea, 1903, pp. 728-729.

The Electrolabyrinthogram

I. Records from the Ventromedial Wall of the Tympanic Cavity of Cats

M. SPECTOR, M.D.; E. G. SZEKELY, Ph.D., and E. A. SPIEGEL, M.D., Philadelphia

Methods for the study of electrical discharges of the brain and of the retina have been developed to such a degree that they are applied as clinical tests. Electrical discharges from the vestibular nerve have been studied in animals in which the electrodes were inserted into the inner ear or placed directly on parts of its nervous apparatus. Spiegel, Wycis, and Umlauf [8] attempted in man to record the electrolabyrinthogram from parts of the external ear, particularly by meatal-retromastoid electrodes. In patients with unilateral eighth nerve tumors these authors compared the effects of rotation upon the normal and upon the affected side. At least in some instances, they found a postrotatory increase of the amplitude of the discharges on the side of the intact labyrinth or intact eighth nerve, as compared with the side of the tumor.

In the course of these studies it became evident that it would be desirable to supplement the clinical studies by animal experiments, in which not only the technical problems of electrode types and mode of their application could be studied, but also the elimination of various extralabyrinthine factors could be performed. The first part of these studies is presented in this communication.

Material and Methods

The experiments were performed on 112 cats. The preliminary preparations were done under intraperitoneal pentobarbital (Nembutal) anesthesia (25 to 30 mg. per kilogram). The approach to the ventromedial wall of the tympanic cavity was similar to that described by de Kleijn.[4] A longitudinal 2.5-3.5 cm. incision was made over the tympanic bulla on the ventral surface of the neck. The site for the incision was located by palpating for the tympanic bulla medial to the angle of the jaw. By blunt dissection lateral to the external maxillary vein one went into the fissure between the submaxillary gland and the digastric muscle to the bulla. The periosteum of the ventral wall of the bulla was incised, peeled back, and the bony bulla was exposed. A Jansen mastoid retractor was inserted. The bony bulla was opened by chisel or burr, and the opening enlarged by rongeur. The tympanic partition dividing the middle ear was left intact. A piece of absorbable gelatin sponge (Gelfoam) was inserted over the round window membrane for protection against injury.

The electric discharges from the ventromedial wall of the tympanic cavity were led off by bipolar (occasionally monopolar) electrodes. Many of the various types of electrodes applied to the surface of the bone failed. Platinum foil electrodes, disks, and spring electrodes gave inconsequential results. A nylon screw with a central passageway for monopolar or bipolar electrodes applied through the bulla to the round window region gave fairly good results. A flat disk electrode cemented into a shallow depression in the bone or affixed with dental amalgam did not hold well. Eventually the following technique was developed.

The mucosa over the petrous bone posterior to the round window was stripped away. About 3 or 4 mm. posterior to the round window a groove was made in the ventromedial wall of the tympanum to a depth of about 1.5 to 2 mm. with a 0.5 mm. cutting burr. The endosteum was not penetrated. About 2 or 3 mm. medial to this opening another sinus was made in the petrous bone. A small silver ball (0.8 to 1 mm. in diameter) electrode was inserted into each groove and tapped down with a dental tamper. Also stainless steel balls soldered to the bare end of insulated silver wire were used. The wire was sheathed by polyethylene tubing. Dental cement was applied above the ball electrode at the entrance of the groove for fixation. The wires were cemented to

Submitted for publication July 28, 1958.

From the Departments of Experimental Neurology and Otorhinology of Temple University School of Medicine and Hospital. Aided by Grants No. B-470 of the National Institute of Neurological Diseases and Blindness, National Institutes of Health, Department of Health, Education, and Welfare, and No. AF 41 (657)-210 of the Wright Air Development Center.

the edge of the bulla or fixed to the neighboring soft tissue. Through a trocar the wires were led subcutaneously to the dorsal part of the neck to protect the wires for the later recording. The skin incision was closed by silk sutures. After an interval of one or more days vestibular tests were done.

Under barbiturate anesthesia the cat gave poor nystagmus responses to turning, so light ether was used during these tests. Cats in a state of catatonia induced by bulbocapnine (20-25 mg/kg.) gave fair responses.

Nystagmus was recorded by leading off the corneoretinal potentials. A clip electrode was attached to the external canthus of each eye. One blade of a clip grasped the skin surface and the other blade the conjunctiva. An indifferent electrode was attached to the auricle. A Grass model III D ink-writing electroencephalograph served as recording apparatus. Average resistance between the bipolar tympanic electrodes was 20,000 ohms.

For the recording of the potentials and labyrinthine stimulation the cat was strapped down in the prone position, with the head erect, on a board that could be rotated on a vertical axis. The electrode wires were affixed by adhesive to the support for the head holder and then threaded through a ring located in the ceiling, above the axis of rotation, thence to the electroencephalograph. In this way recordings could be made while the cat was rotated. Ten rotations were made in 20 seconds. Caloric stimulation was performed by instillation of 20 to 50 cc. of ice water into the external auditory canal by means of a cannula (Gauge 15) covered with polyethylene tubing and affixed to the pinna by a clamp.

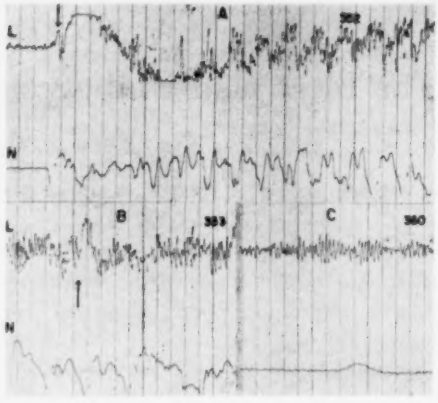
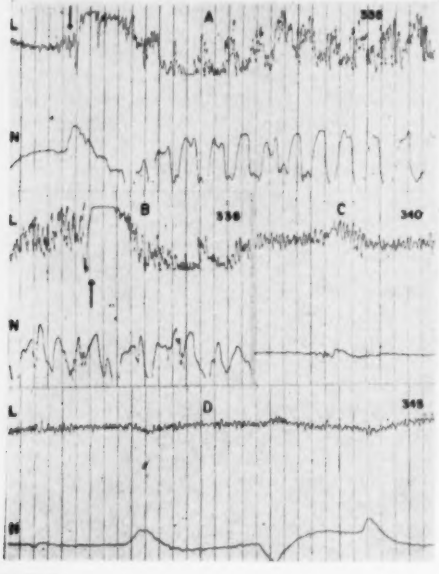


Fig. 2 (Cat No. 62).—Effect of rotation on left-sided labyrinthogram (*L*) after ligation of the carotids. Rotation to right starts at ↓ (Graph *A*), ends at ↑ (Graph *B*). Postrotatory reaction in *B* and *C*. Between *B* and *C* interval of 35 seconds. *N*=nystagmogram. Time intervals: ⅕ second.

Fig. 1 (Cat No. 62).—Effect of rotation on left-sided labyrinthogram (*L*). Rotation to right starts at ↓ (Graph *A*), ends at ↑ (Graph *B*). Postrotatory reaction in *B* and *C*. Between *B* and *C* interval of 20 seconds. *D*=45 seconds post-rotation. *N*=nystagmogram, time intervals (between vertical lines): ⅕ second.



Results

Records of the potentials picked up from the ventromedial wall of the tympanum in the resting stage and during and after labyrinthine stimulation showed that such a stimulation increased the amplitude of these potentials. This could be observed during as well as after arrest of the rotation (Figs. 1-3) and also after caloric stimulation (Fig. 4). It is probable that there were also changes in the frequency associated with the changes in the amplitude; however, a definite statement in this respect seems to be hazardous since it could be based only on records of single unit preparations as, for instance, those published by Lowenstein.[5] *

* Definite differences between the effect of clockwise and counterclockwise rotation could not be found, probably because the tympanic electrodes picked up discharges not only from the ampullae of the horizontal canals but also from other labyrinthine receptors.

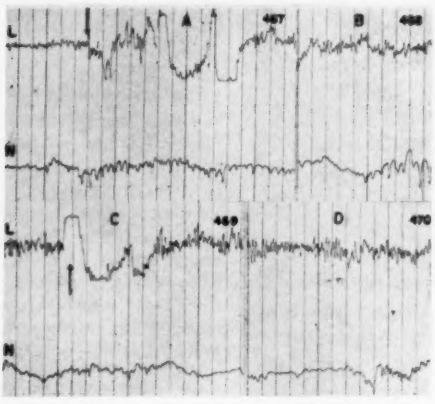


Fig. 3 (Cat No. 62).—Effect of rotation on left-sided labyrinthogram (*L*) after decerebration. Rotation to right starts at ↓ (Graph *A*), ends at ↑ (Graph *C*). Reaction during rotation in *A* and *B*, and 10 seconds post rotation in *D*. Between *A* and *B* and *C* and *D* intervals of 5 seconds. *N*=nystagmogram. Time intervals: ½ second.

Besides such a continuous type of reaction, a second type could be observed consisting in the rhythmic appearance of bursts of medium to high-amplitude discharges. These rhythmic bursts appeared after a latent period of 15 to 25 seconds, following the end of the caloric stimulation (Fig. 4) and 7 to 10 seconds after cessation of rotational stimuli (Fig. 5). Their appearance sometimes coincided with that of the electrographically recorded nystagmic eye movements, or each rhythmic burst preceded such a nystagmic movement. Each burst lasted about 0.2 to 0.5 seconds. While a certain degree of synchronism between the appearance of the nystagmic movements and the rhythmic bursts could be observed in some electrolabyrinthograms, there were also instances in which the two phenomena appeared independently of each other, or in which the rhythmic discharges in the electrolabyrinthogram outlasted the recorded nystagmic movements. The increase of the amplitude continued for 25 to 40 seconds and the rhythmic bursts for 35 to 90 seconds.

The question arises how much of this reaction originated in the labyrinth and how much was extralabyrinthine in origin. After injection of cocaine solutions into the inner ear through the round window, it was found that after injection of a 1% solution a slight increase of potentials was still noticeable during rotation but no longer after rotation. After injection of a 4% solution, the post-rotatory increase of potentials was also abolished (Fig. 6). Furthermore, the following control experiments were performed: (1) decerebration with removal of the brain cranial from the section that passed through the mesencephalon; (2) curarization; (3) combination of decerebration and curarization. Elimination of the parts of the

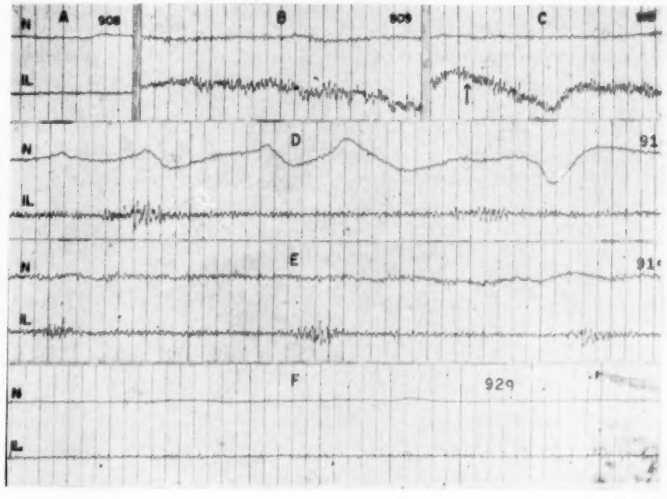


Fig. 4 (Cat No. 95).—Effect of stimulation with ice water on left-sided labyrinthogram (*IL*). *A*=before and *B*=during cold water instillation in the left external meatus. Instillation ends at ↑ (Graph *C*). Reaction 25 seconds (*D*), 40 seconds (*E*) and 90 seconds (*F*) after instillation. N= nystagmogram. Time intervals ⅒ second.

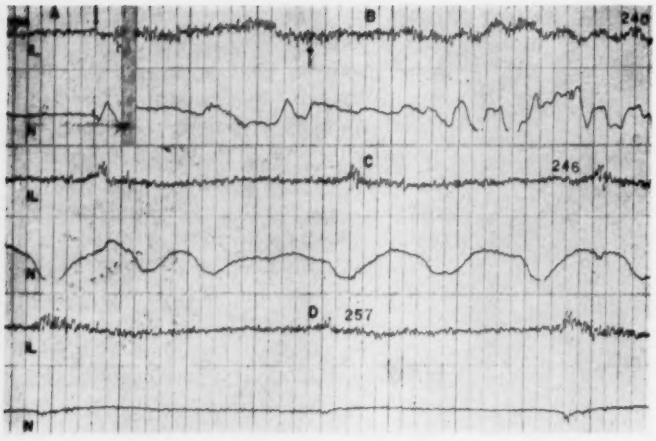


Fig. 5 (Cat No. 59).—Effect of rotation on left-sided labyrinthogram (*IL*). Rotation to left starts at ↓ (Graph *A*), ends at ↑ (Graph *B*). Between *A* and *B* interval of 20 seconds. Postrotatory reaction in *C* (25 seconds after rotation) and *D* (45 seconds after rotation). *N*=nystagmogram. Time intervals: 1/10 second.

cerebrum cranial to the mesencephalon (Fig. 3), as well as of the muscle potentials by curarization and also a combination of these procedures (Fig. 7) did not prevent the above described changes of potentials picked up from the ventromedial wall of the tympanic cavity on or after labyrinthine stimulation.

However, one had also to consider the possibility that the tympanic electrodes may pick up the electric discharges not only from the inner ear, but also from the medulla oblongata, particularly from the vestibular nuclei. Therefore, in a fourth series of experiments in decerebrated and curarized cats the opposite labyrinth was paralyzed by

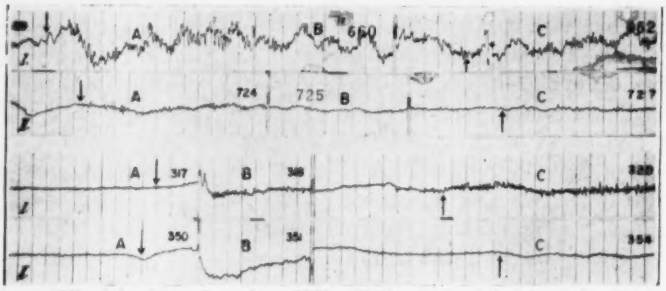


Fig. 6 (Upper records, Cat No. 35).—Effect of rotation on right-sided labyrinthogram before (*I*) and 43 minutes after (*II*) injection of cocaine HCl (1%) in the right labyrinth through the round window. Rotation to left starts at ↓ (Graph *A*), ends at ↑ (Graph *C*). Reaction during rotation in Graph *B*. (Lower records, Cat No. 33).—Left-sided labyrinthogram before (*I*) and 10 minutes after (*II*) injection of cocaine HCl (4%) in the left labyrinth. Rotation to right. Time intervals: 1/5 second.

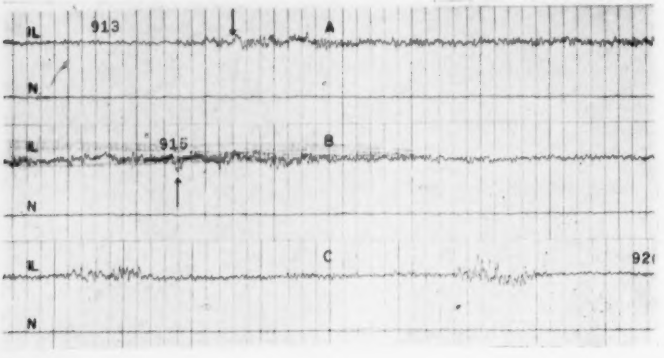


Fig. 7 (Cat No. 82).—Effect of stimulation with ice water on left-sided labyrinthogram (*IL*) after curarization and decerebration. Cold water instillation in the left external meatus starts at ↓ (Graph *A*), ends at ↑ (Graph *B*). Reaction 25 seconds after instillation in Graph *C*. *N*=nystagmogram. No nystagmus recorded because decerebration also cut motor nerves to the eye muscles. Time intervals: 1/10 second.

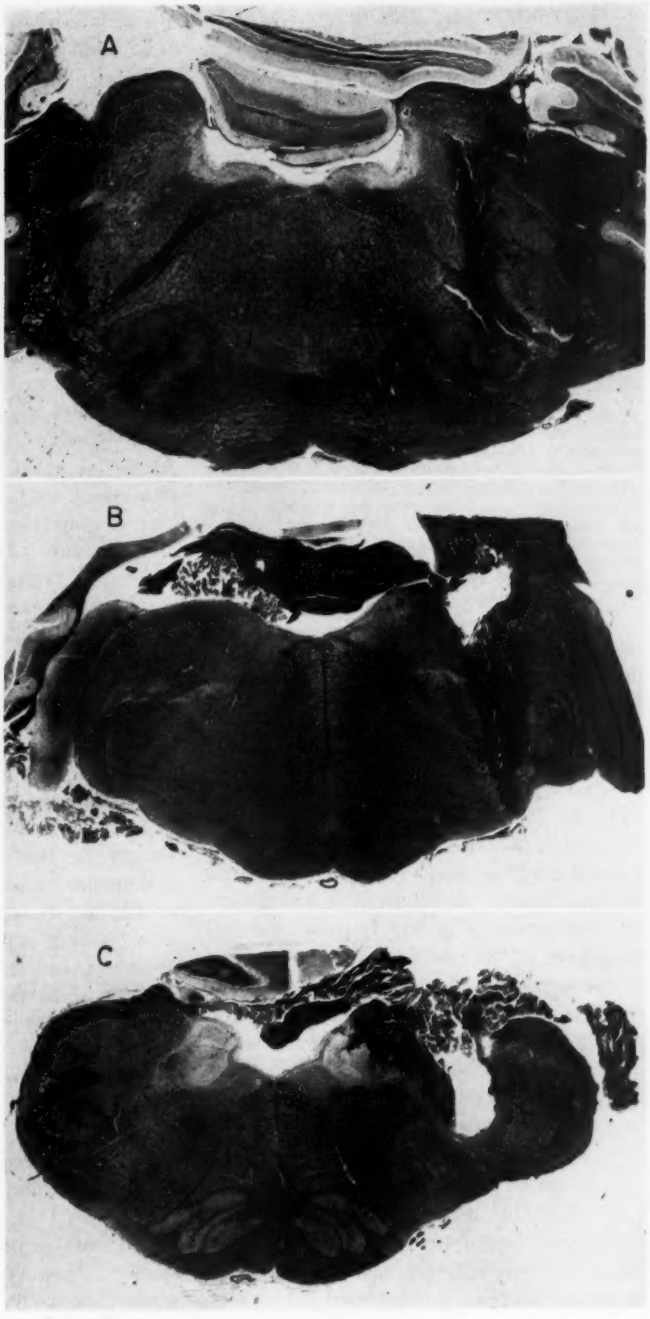


Fig. 8 (Cat No. 112).—Incision into the pons and medulla oblongata of the left side. At the level of the knee of the root of the facial nerve (*A*), the lesion extends from the brachium conjunctivum to the superior olive, severing the vestibular fibers before they enter the vestibular nuclei. In the most cranial levels of the oblongata (*B*), the dorsal part of the lesion destroys the descending root of the eighth and extends through the reticular formation, medial from the substantia gelatinosa V to the base. The most caudal part of the lesion (*C*) destroys the most caudal part of the descending root of the eighth and the medial part of the cuneate nucleus, and reaches the lateral part of the nucleus dorsalis X; it extends ventrally to the reticular formation adjacent medially to the substantia gelatinosa V.

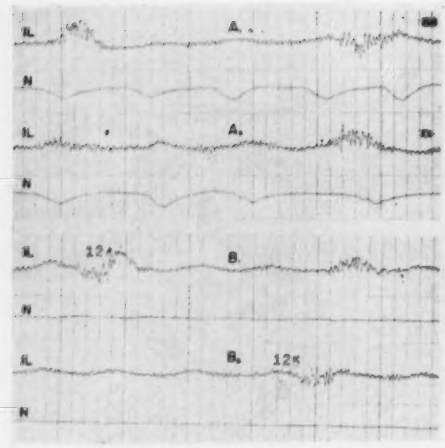


Fig. 9 (Cat No. 112).—Effect of left-sided stimulation with ice water on left-sided labyrinthogram (*IL*). A_1 and A_2=after 4% cocaine injection in right labyrinth. Reaction 25 seconds after cold water instillation. B_1 and B_2=after additional incision into the pons and medulla of the left side. Record 20 seconds after ice water instillation in the left external meatus. *N*=nystagmogram. No nystagmus in B_1 and B_2 on account of elimination of right labyrinth and of impulses from the left vestibular nuclei. Time intervals: 1/10 second.

cocainization and incisions were made along the inner aspect of the restiform body homolateral to the side of the recording electrodes in order to cut the vestibular fibers entering the vestibular nuclei and/or to destroy cell groups of these nuclei (Fig. 8). These lesions did not prevent the appearance of electrical discharges of increased amplitude and of the above-described rhythmic bursts on labyrinthine stimulation by calorization (Fig. 9). It seems justified therefore to ascribe the above-described phenomena chiefly to labyrinthine discharges, although muscular and cerebral potentials may sometimes be superimposed upon the electrolabyrinthogram. The relationship to cochlear potentials will be studied in a subsequent paper.

Comment

It could be demonstrated that electric discharges elicited by rotational or caloric stimuli and originating in the labyrinth could be picked up from the ventromedial wall of the tympanic cavity of cats, although artifacts induced by cerebral and muscular potentials may sometimes interfere. Of special interest seems to be the finding that these discharges may appear not only continuously but in some instances also in rhythmic bursts.

It is known that the nystagmus of labyrinthine type showing a slow and fast component may be observed also in spite of the absence of both labyrinths as shown by Bechterew's [1] compensatory nystagmus that appears if first one labyrinth has been eliminated and after compensation of the nystagmus directed to the second labyrinth this is also extirpated. The finding (Spiegel and Sato [7]) that in bilaterally labyrinthectomized cats a nystagmus can be produced by a lesion of the vestibular nuclei, producing an imbalance between the left- and right-sided nuclei, also indicates the existence of central mechanisms able to innervate a nystagmus with the characteristics of a vestibular nystagmus. However, these experiments do not exclude the possibility of peripheral, labyrinthine factors in the mechanism of the rhythm of the vestibular nystagmus. Maupetit [6] has expressed the hypothetical view that the quick phase of the nystagmus is a labyrinthine reflex. An exclusive origin of the rhythm in the peripheral labyrinth is refuted by the above-mentioned experiments. Against Maupetit's theory, Dusser de Barenne [3] objects also that on habituation to rotation, the quick phase of nystagmus disappears gradually, while the slow phase, expressing itself as a slow deviation, remains (Dodge [2]). The present findings show that a peripheral mechanism in the labyrinth may also play a part.

However, it should be emphasized that rhythm of the bursts in the electrolabyrinthogram frequently does not coincide with that of the nystagmus. Often no eye movements could be recorded that would correspond to the labyrinthine bursts, or vice versa (Fig. 4). This may be due partly to an insufficient sensitivity of the recording system. However, in view of these discrepancies, the part played by the peripheral labyrinth in originating the rhythm of

nystagmic discharges should not be overestimated.

Summary

By means of electrodes applied to the ventromedial wall of the tympanic cavity in cats electrical potentials were picked up that were increased by labyrinthine stimulation (rotation and caloric stimulation). These reactions persisted after decerebration, paralysis of muscles by curare, elimination of the opposite labyrinth and of impulses entering the homolateral vestibular nuclei. They were interpreted as discharges chiefly of the nonacoustic part of the inner ear. The electrolabyrinthogram following labyrinthine stimulation showed the rhythmic appearance of bursts which were to a certain extent independent of the rhythm of nystagmus. The relationship of the electrolabyrinthogram to cochlear potentials will be studied in a subsequent paper.

3400 N. Broad St. (40).

REFERENCES

1. Bechterew, W.: Ergebnisse der Durchschneidung des N. acusticus, nebst Erörterung der Bedeutung der semicirculären Canale für das Körpergleichgewicht, Arch. ges. Physiol. 30:312-347, 1883.

2. Dodge, R.: Habituation to Rotation, J. Exper. Psychol. 6:1-35, 1923.

3. Dusser de Barenne, J. G.: The Labyrinthine and Postural Mechanisms, in Handbook of General Experimental Psychology, edited by C. A. Murchison, Worcester, Mass., Clark University Press, 1934, pp. 204-246.

4. de Kleijn, A.: Zur Technik der Labyrinthextirpation und Labyrinthausschaltung bei Katzen, Arch. ges. Physiol. 145:549-556, 1912.

5. Lowenstein, P.: Peripheral Mechanisms of Equilibrium, Brit. M. Bull. 12:114-118, 1956.

6. Maupetit, R. J. A.: Étude clinique sur le nystagmus rhythmique provoqué, Thèse méd., Bordeaux, 1908.

7. Spiegel, E. A., and Sato, G.: Experimentellstudien am Nervensystem: Über den Erregungszustand der medullären Zentren nach doppelseitiger Labyrinthausschaltung, Arch. ges. Physiol. 215: 106-119, 1926.

8. Spiegel, E. A.; Wycis, H. T., and Umlauf, Ch.: Attempts to Record the Human Electrolabyrinthogram, Fed. Proc. 9:119, 1950.

Postcricoid Carcinoma and Obstructing Lesions of the Thoracic Esophagus

A New Operation for Replacement of the Esophagus

HENRY J. HEIMLICH, M.D., New Rochelle, N. Y.

Introduction

An operation will be presented which enables the entire esophagus to be replaced, from the pharynx to the cardia of the stomach. The procedure has been used successfully in the treatment of benign and malignant obstructions of the esophagus. Gastrointestinal continuity is restored physiologically. The patient in whom the entire esophagus has been resected or by-passed by a reversed gastric tube can eat all foods in a normal manner.

The operation will be described and three case reports presented which illustrate its use in the treatment of benign and malignant esophageal obstructions. I have performed this operation in 10 consecutive patients, 2 of whom will be presented. The first case demonstrates the successful treatment of carcinoma of the upper thoracic esophagus. The second report describes the restoration of gastrointestinal continuity in a patient with a lye stricture of the esophagus.

I described the reversed gastric tube operation in 1955, and, subsequently, was informed by Dr. Dan Gavriliu of Bucharest, Romania, that he had been performing this operation since 1951. In 1956, at the invitation of the Romanian Academy of Medical Science, I spent a week in Bucharest with Dr. Gavriliu. The third case report is that of a patient of Dr. Gavriliu; it illustrates a successful replacement of the esophagus in a patient with postcricoid carcinoma.

Submitted for publication July 11, 1958.

Presented at the New York Academy of Medicine Section on Otolaryngology, April 16, 1958.

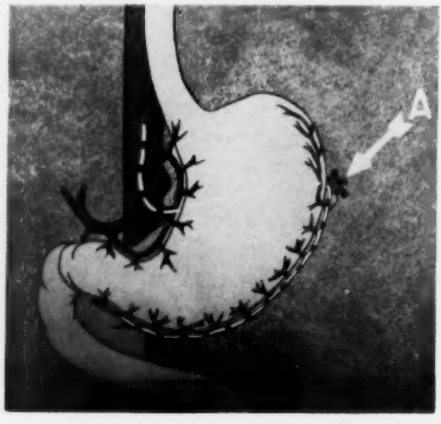
Adjunct Thoracic Surgeon, Montefiore Hospital, and Assistant Clinical Professor of Surgery, New York Medical College.

The Operation

The abdomen is entered through a left subcostal incision. The tail of the pancreas is freed from its bed. The splenic vessels are ligated in the hilus of the spleen, preserving the origin of the left gastroepiploic vessels, and the spleen is removed. The blood passing through the splenic vessels is now directed into the left gastroepiploic vessels, which supply the greater curvature of the stomach (Fig. 1A).

An incision is made into the greater curvature of the stomach through the anterior and posterior walls of the antrum, approximately 5 cm. proximal to the pylo-

Fig. 1A.—The spleen has been resected. (*A*) ligated splenic vessels. The dash line indicates the blood flow directed from the splenic vessels into the left gastroepiploic vessels to supply the greater curvature of the stomach.



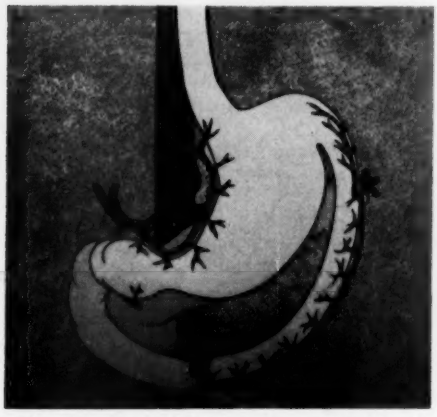


Fig. 1B.—An incision has been made into the antrum and extended parallel to the greater curvature. The gastric tube thereby formed remains attached to the stomach at the fundus.

rus. The incision is extended parallel to the greater curvature, at a distance of 2 cm., through both walls of the stomach, to a point high on the fundus (Fig. 1B). The incised edges of the anterior and posterior walls of the stomach are sutured together, forming the gastric tube and reconstituting the remainder of the stomach (Fig. 2A).

An incision is made in the neck anterior and parallel to the left sternomastoid muscle, extending upward from the suprasternal notch. A subcutaneous tunnel is created in the avascular plane anterior to the sternum, extending from the xiphoid to the incision in the neck. The gastric tube is drawn through the tunnel and is brought high in the neck, accompanied by its vascular supply, the left gastroepiploic vessels. The freed tail of the pancreas extends to the costal margin. The pancreas is accompanied by the splenic vessels from which the left gastroepiploic vessels originate. Thus, the vascular supply of the gastric tube is long enough to reach to the neck (Fig. 2B).

The cervical esophagus is dissected free and transected approximately 2 cm. distal to the pharynx. The distal end of the cervical esophagus is closed and is allowed to retract into the superior thorax. The reversed gastric tube is anastomosed end-to-end to the proximal cervical esophagus, restoring gastrointestinal continuity (Figs. 3 and 4).

The functions of the esophagus can now be performed by the reversed gastric tube. If the esophageal obstruction is caused by a benign stricture or has been proved to be an inoperable carcinoma, it is not necessary

Fig. 2A.—The gastric tube has been reversed and lies on the chest. The anterior and posterior walls of the stomach have been approximated, completing the gastric tube and reconstituting the stomach. A rubber catheter is seen emerging from the gastric tube. This catheter has been used as a mold on which the tube is constructed and it is removed at this time. The tail of the pancreas (*A*) has been freed, and carries the splenic vessels to the costal margin. This permits the gastroepiploic vessels (*B*) to accompany the gastric tube to the neck.

Figure 2A

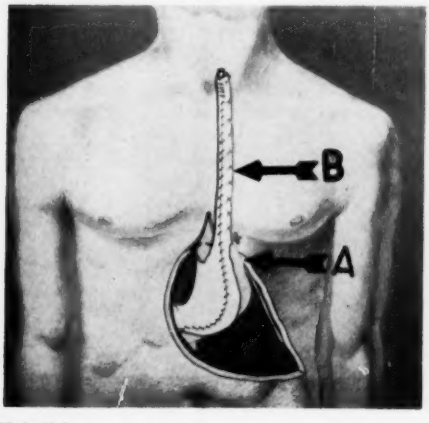
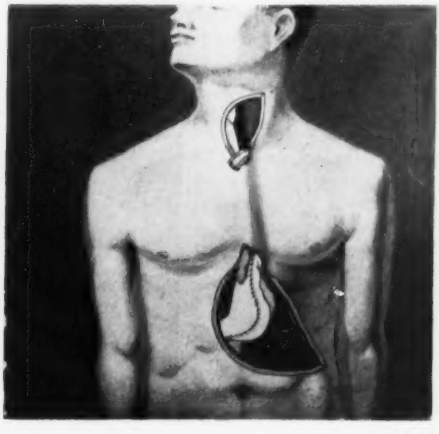


Fig. 2B.—The reversed gastric tube has been drawn through a subcutaneous tunnel, to the neck.

Figure 2B



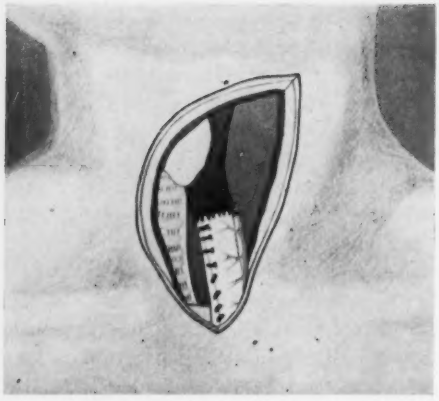


Fig. 3.—Through the incision in the neck, the cervical esophagus has been divided. Its distal end has been closed and has retracted into the thorax. The proximal end of the esophagus has been anastomosed end-to-end to the reversed gastric tube. The gastroepiploic vessels are seen accompanying the gastric tube.

to enter the chest. If a resectable carcinoma is present, a right thoracotomy incision is made, and the entire esophagus is resected. I have preferred to perform the thoracotomy at a second stage, 10 days to two weeks postoperatively.

Report of Cases

CASE 1 (Montefiore Hospital).—*Present Illness.* This 35-year-old woman was unable to eat soft foods for 15 days prior to admission on Aug. 26, 1957, but could swallow liquids. She had lost 20 lb. in the previous three months.

Esophagram, Aug. 20, 1957.—Partial obstruction in the upper third of the esophagus (Fig. 5A).

Esophagoscopy, Sept. 10, 1957.—A constricting irregular lesion partially obstructed the upper third of the esophagus. Biopsy: Epidermoid carcinoma.

Operation, Sept. 12, 1957.—A reversed gastric tube was constructed and anastomosed to the cervical esophagus.

Course.—The postoperative course was uneventful.

Operation, Sept. 23, 1957.—A right thoracotomy was performed. A firm lesion was present in the esophagus beneath the arch of the azygos vein. There was no evidence of spread of the carcinoma outside of the lumen. The entire esophagus, from the previously closed upper end to the cardia of the stomach, was excised with surrounding lymph nodes, none of which were enlarged. Microscopically, all nodes were free of tumor. The opening into the cardia was closed by inversion (Fig. 5B).

Course.—There was no postoperative difficulty. A superficial wound infection developed in the thoracotomy incision and healed rapidly after drainage. Fluids were taken by mouth on the fourth postoperative day, and soft foods on the sixth postoperative day. On the eighth postoperative day she was given a regular diet and has continued to eat all foods at home. She has gained 13 lb. since operation and at present is completely well (April, 1958) (Figs. 6A and B).

CASE 2 (Metropolitan Hospital).—*Present Illness.*—This 45-year-old woman had been unable to swallow solids or liquids since swallowing lye in a suicide attempt on May 7, 1956. A gastrostomy had been performed at the time of injury, and she had been nourished by means of a gastrostomy tube for one and one-half years. The patient had been admitted to this hospital in June, 1957, at which time an exploratory laparotomy was performed with the intention of constructing a reversed gastric tube. However, after the splenectomy, it was felt that the left gastroepiploic vessels had been inadvertently injured. Therefore, because it was known that under these circumstances a gastric tube will necrose, the operation was not performed and the patient was discharged. The patient was readmitted on Jan. 2, 1958. It was felt that the reversed gastric tube procedure could be performed in the event that the gastroepiploic vessel had developed

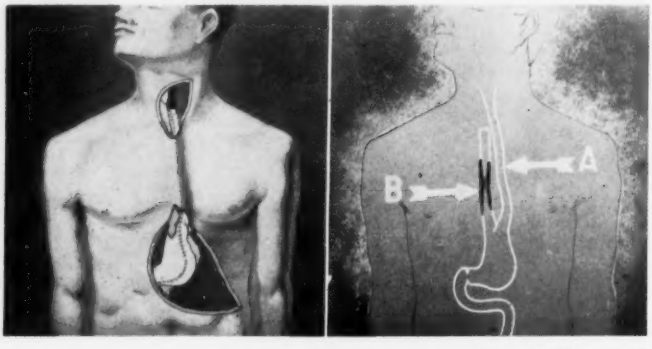


Fig. 4.—Completed Operation. The functions of the esophagus can now be performed by the reversed gastric tube (*A*). The diseased esophagus (*B*) is nonfunctioning. It can be resected if an operable carcinoma is present; otherwise, it is left in situ.

Fig. 5A (Case 1).—Preoperative esophagram, Aug. 20, 1957, demonstrating partial obstruction due to carcinoma (*C*).

Fig. 5B (Case 1).—Resected esophagus. (*C*), the carcinoma. (*D*), the distal end of the cervical esophagus, which was closed at the time the reversed gastric tube was constructed.

collaterals; otherwise, another means of esophageal replacement was contemplated.

Esophagram, May 29, 1957.—Marked narrowing of the esophagus below the level of the fifth thoracic vertebra with almost complete obstruction. The esophagus above this level is tremendously dilated (Fig. 7A).

Operation, Jan. 31, 1958.—Exploratory laparotomy: The left gastroepiploic vessels were adequate, and a reversed gastric tube was constructed and anastomosed to the cervical esophagus. Although the stomach was markedly thickened and somewhat fibrotic, there was no difficulty in creating a gastric tube of adequate length to reach to the neck. The fact that the left gastroepiploic vessels were adequate may have been due to the development of collaterals. It is more likely that the experience which I had gained in performing eight reversed gastric tube operations in the six-month interval since the first procedure on this patient, enabled me to evaluate more accurately the status of the vascular supply.

Course.—A small salivary fistula which developed in the neck healed spontaneously in several weeks. During this period the patient received nourishment by means of gastrostomy feedings. At the present time (April, 1958), she eats a regular diet, and has gained 6 lb. since the reversed gastric tube operation (Fig. 7C).

CASE 3.—*Present Illness.*—This 52-year-old man had a squamous-cell carcinoma of the cervical esophagus with invasion of the trachea, larynx, and thyroid.

Operation, July, 1952.—A gastrostomy was performed, which was followed two weeks later, by a radical neck dissection and resection of the larynx, cervical esophagus, and thyroid.

Operation, November, 1953.—A gastric tube esophagoplasty was performed by Dr. Dan Gavriliu in Bucharest, Romania.

Course.—The patient has been able to eat solid foods since the 10th postoperative day. He had no evidence of recurrence of the carcinoma when I saw him in October, 1956, and had developed a clear esophageal voice through the gastric tube (Fig. 8).

Fig. 6A (Case 1).—Postoperative view of a 35-year-old woman who had a reversed gastric tube operation followed by a total esophagectomy for carcinoma of the upper third of the thoracic esophagus.

Summary and Conclusions

In 10 patients with benign and malignant esophageal obstructions, the esophagus has been replaced with a reversed gastric tube. The reversed gastric tube operation provides a physiological means of esophageal replacement and offers the following advantages:

1. The reversed gastric tube looks and functions like a normal esophagus.
2. All foods can be swallowed in a normal manner.
3. The entire esophagus can be replaced.
4. Eighty per cent of the stomach remains in the abdomen, retaining the normal storage function.
5. Peptic esophagitis does not occur because the antrum, which does not secrete acid, forms the portion of the reversed gastric tube which is anastomosed to the esophagus.
6. There is no intestinal organ or foreign material interposed between the proximal esophagus and the stomach.

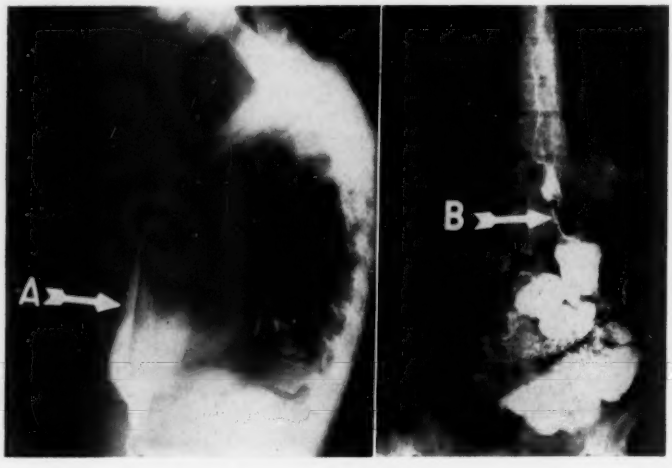


Fig. 6B (Case 1).—Barium swallow through the reversed gastric tube. (*A*), the gastric tube, anterior to the sternum. (*B*), reversed gastric tube emptying into the residual stomach.

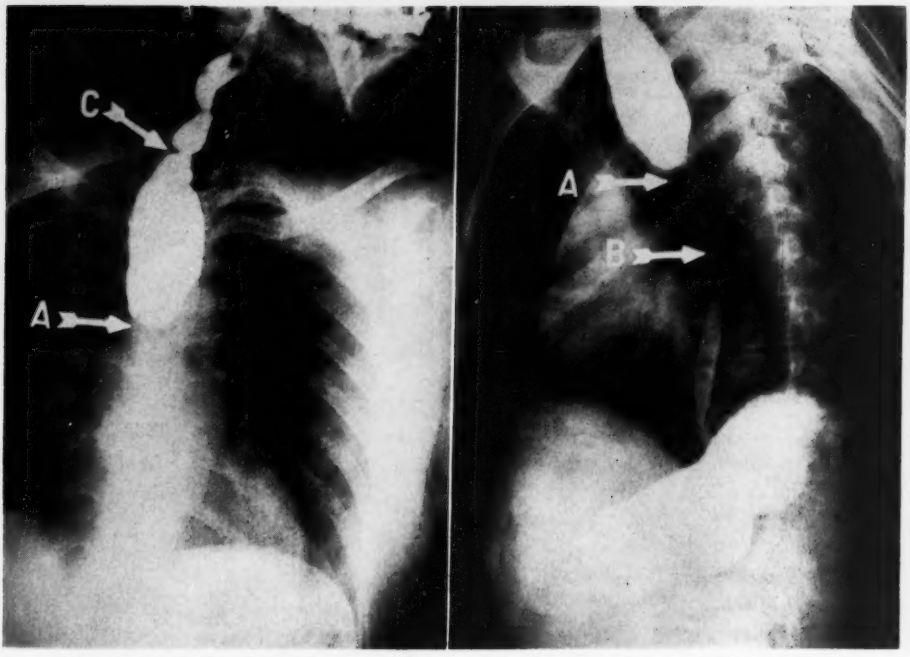


Fig. 7A (Case 2).—Preoperative esophagram, May 29, 1957. An almost complete obstruction (*A*) is demonstrated at the level of the aortic arch. A thin stream of barium is seen passing through the strictured esophagus (*B*). Note the higher partial stricture (*C*). It is not unusual to have two obstructions develop following the swallowing of lye. It is important that the anastomosis of a substitute esophagus be made above the highest stricture.

Fig. 7B (Case 2).—Barium swallow through the reversed gastric tube (*D*). In the lateral film, the antesternal position of the gastric tube is evident. The anastomosis between the esophagus and the gastric tube (*AA*) is higher than the uppermost stricture (*C*) seen in Figure 7A. The barium which was swallowed has flowed up into the strictured esophagus (*B*), which remains in situ with its upper end closed.

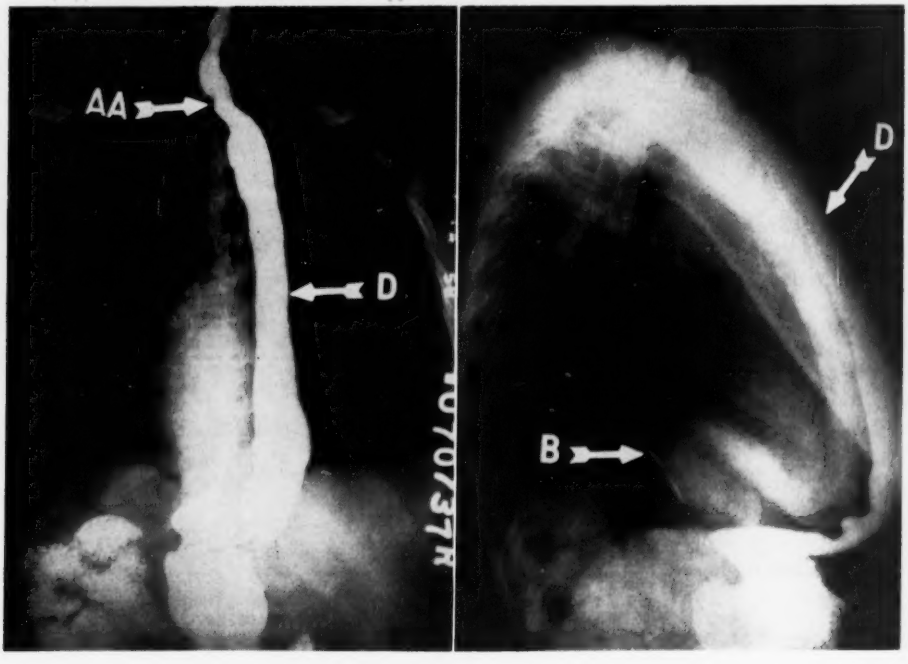


Fig. 7C (Case 2).—Postoperative view of the patient, illustrating the appearance of the reversed gastric tube which lies anterior to the sternum.

7. The blood supply to the reversed gastric tube is excellent.

8. The reversed gastric tube is brought to the neck through a subcutaneous tunnel. If the esophageal obstruction is caused by a benign stricture or an inoperable carcinoma, a thoracotomy need not be performed.

Figures 1, 2, 3, and 4 are from the motion picture "Esophageal Replacement with a Reversed Gastric Tube." This film was supported by a grant-in-aid from the Baxter Laboratories.

140 Lockwood Ave.

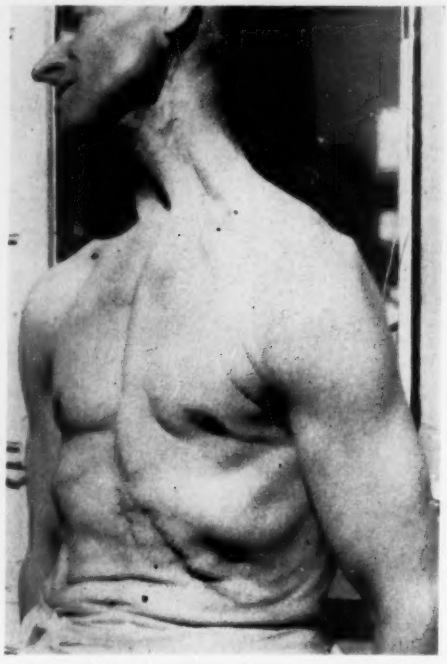


Fig. 8 (Case 3).—Man, 52 years old, who had a resection of the cervical esophagus, trachea, larynx, and thyroid for invasive squamous-cell carcinoma. The patient is seen three years after resection. He eats a regular diet and has developed a clear esophageal voice through the gastric tube.

Pathology and Therapy of the Singing Voice

PAUL J. MOSES, M.D., San Francisco

Introduction

A discussion of the pathology and therapy of the singing voice is a most timely subject. In the last decade Husson has presented completely new theories on the physiology of phonation, theories so far not sufficiently evaluated as to their impact on what we know or thought to know about singing. New endocrinological ideas are changing our basic concepts about the variability and pathology of the singing voice. Newly gained psychological insight, also, into the singer's personality makes it evident that the laryngologist faces a changing situation which he has to master if he attempts to treat singers at all properly.

It has not been easy for the laryngologist to become interested and trained in phoniatrics. In the past he has seen singers only for acute upper respiratory infections, in an emergency situation to enable the artist to sing a certain performance. Too often, not until a singer enters his office does the physician change into a voice specialist, with insight into the organic condition of vocal organs, but without any knowledge of the functions of singing. But now, with the increasing number of singers in this country, through newly formed operatic enterprises and the many performers used in the different branches of mass communication such as movies, radio, and television, the responsibilities of the laryngologist are growing so as to become not only medical but cultural tasks.

Submitted for publication July 17, 1958.

Associate Clinical Professor, Division of Oto-Laryngology (Speech and Voice Section), Stanford University School of Medicine.

Presented at the Annual Session of the California Medical Association, Section on Ear, Nose and Throat, Los Angeles, April 28, 1958.

The Singer as Patient

Are singers different from other patients? They are often believed to be temperamental, superstitious persons, full of fears and anxieties, demanding special attention from their physicians. And yet the physician often does not know what it means to be in perfect voice at a certain minute, to be forced to recreate one's instrument, "voice," every day, exactly contrary to the pianist, who finds his instrument ready and well tuned before him on the stage. The emotional situation of the vocal artist before a performance has to be evaluated instead of being ridiculed as nervousness. Otherwise the therapist cannot differentiate between true rhinitis and the psychosomatic swelling of nasal turbinates that indicates anxiety.[1]

Too often laryngologists have only professional contact with the singer as a patient during disease. They do not attend concerts regularly, nor have they ever watched demonstration lessons given by reputable singing masters in order to learn what it means to train a voice and which functions of the voice are trainable. The doctor might also recall that the examination of the larynx would be impossible if the singing master, Manuel Garcia, had not invented the larynx mirror, which we still use without feeling too grateful to that nonmedical genius.

Diagnostic Problems

Visual methods are most imperfect for the establishment of vocal diagnosis. We see the cords while the tongue is stretched out, with the larynx in the highest possible position. In longitudinal direction this means that the larynx is placed about one inch higher than it would be placed in singing. The trachea is stretched accordingly to an

unphysiological position, which certainly does not enable us to inform the singer about the closing function of the glottis. The vowel EE, which the patient produces, results from vibrations of the inner fibers of the cords on the highest falsetto tone, a coordination not used in normal vocal performance. The stroboscope, which would make these vibrations of isolated parts of the cords visible, is hard to obtain and still reveals functions in unnatural placement of the larynx. One therefore depends on visual as well as acoustic impressions to establish a diagnosis.

If the singer asks in normal speaking voice whether his cords close—his eternal nightmare—the voice already indicates whether air escapes between incompletely closed vocal cords. The simplest test is to let the patient vocalize against a tissue paper, which does not move as long as no so-called wild air escapes through a perfectly closed glottis. Another standard question of the singer, "Are my cords red or white?" is often hard to answer factually and interpret since many female singers who perform daily reveal very small telengiectasis or petechial hemorrhages during the menstrual period. Bassos and baritones often have yellowish cords.

Such problems of peripheral laryngeal pathology as polyps and pachydermic changes do not have to be mentioned here, since they also affect the speaking voice. The problem of the singers' node is of increasing concern to the voice specialist, not so much in singers of classical music but mainly in the female popular singer. Vocal styles change with the changes in vocal and sex appeal. The present style suppresses the head voice in girls; the chest voice is pressed upward, and the resulting hoarseness characterizes the style since such hyperfunction of intensity, required even with microphones, and the overuse of the chest register make it impossible to keep the cords closed.[2] The result is often the formation of small symmetrical nodes between the anterior and middle third of the cords. Surgical removal is usually contraindicated. Voice rest, which might relieve the condition, is senseless because to resume the same way of singing will cause the nodes to return within days.

The changes, too, in operatic singing have created a new situation for the laryngologist. The task of the singer during the 17th and 18th centuries was "bel canto," with emphasis on technical perfection. In the last century the singing of songs and lieder required a personal expression; but, even more, the singing of the works of Verdi and Wagner created new vocal tasks unheard of before, especially in the accompaniment of the large orchestras. In "bel canto" the emphasis was on voice alone. Today the singer has to express the whole personality of the role; acting is as important as singing, so the singer's concentration must be diversified and no longer completely concentrated on voice production. The earlier style gave the singer more oral gratification and more narcissistic outlets. Nowadays perfect vocal ability and musicianship, as well as complete understanding of the role and excellent acting are taken for granted, and this makes it more difficult to perform a part. The strain of frequent traveling, constant change of climate, and short-term contracts add to the singer's vocal anxieties. These must all be taken into account and make the differential diagnosis between functional and organic pathology a most urgent problem for the laryngologist.

Functional Disorders

Functional disturbances have been called phonasthenia for several decades. The term needs to be redefined, since the mechanistic theory on which it is based is outdated, and the part of the word which indicates weakness has become almost as meaningless as the term myasthenia. In fact, a great variety of psychogenic causes are obscured by conservative emphasis on organicity. Contact ulcers and many allergic manifestations of the larynx which impair singing belong to these typical psychosomatic phenomena. The symptoms of phonasthenia are well known:

the intonation is disturbed, the tones poorly sustained; pitch becomes flat or sharp. The vocal intensity is irregular, singing softly becomes increasingly difficult because the cords are either hypo- or hypertensive. The tenor who cannot sing anything but forte and fortissimo is well known. Sounds become disturbed: they can be pressed and throaty, the tongue can move too close to the palate or to the posterior pharyngeal wall (the "dumpling-" or *Knödel-Singer*). Breathy tones are produced with incompletely closed glottis; the wild air escapes.

Tremor of the voice, "tremolo," is produced by an insufficient amount of exhaled air during phonation. Since respiration is disturbed primarily in emotional upsets, one has to differentiate between a neurotic tremolo, a poorly trained voice, or a senile voice.

The theories of Husson have greatly discouraged the overemphasis on correct breathing technique as the basis and guarantee for ideal singing.[3] He believes that speaking, singing, and breathing are only loosely related. Because of the fact that the larynx was originally an organ of respiration and only secondarily of phonation, he rejects the old mechanical theory that exhaled air produces vibrations of the vocal cords. According to his research they result from influxes sent by the recurrent nerve. Phonation is regulated entirely on the cerebral level, not by respiration, which in itself depends on actions in the larynx and the resonator. Changes within the tracheobuccal canal cause respiratory changes in singing.

For the speaking voice the initial stimulus comes from the diencephalon and can even be of bulbar origin. Since in speaking there is no intentional pitch of a tone, there is no integration with auditory centers. In normal conversation pitch does not play an important part, so this integration becomes unnecessary.

But in singing and in certain *recitativo* speaking in opera, as soon as the pitch is fixed the diencephalic stimulus is suppressed and cortical centers take over. The highest perfection is reached through cortical control, a more mechanical one through the diencephalon, and the least expressive through bulbar stimuli. Therefore, when these new theories are applied to the pathology of the singing voice, several differences in management and treatment appear:

1. Since Husson observed vibrations of vocal cords independent of any *respiration*, breathing exercises are of value for singing only when they are performed during phonation. They are worthless if tones are not produced simultaneously.

2. Both *singing and speaking* on pitch stem from the same center. It therefore means an increased strain on singers on the operatic stage to switch from operatic classical singing to conversation without fixed pitch. I have in my own practice observed famous classical singers who cannot perform regularly in musicals. The singing of classical operatic parts does not hurt them as long as the speaking parts are limited to the so-called *recitativo*, which is semispeaking on a fixed pitch for each syllable.

3. *Disturbances* of the voice on the cortical level are limited to singing—a fact often observed, since phonasthenia in singing disappears as soon as the singer speaks, but one never explained before.

Range and pitch and their precise performance depend on precise functioning of the psychoauditive representation of sound. Since pitch is under cortical control, the initial stimulus for the vocal cords is integrated through rhythmic activity of the auditory centers, without stimulation of the auditory nerve.

4. *Range.* A close relationship between the speaking and the singing range has always been taken for granted. This is not so, as the voice specialist has long known. In general one tended to consider any discrepancy between the two ranges as damaging to the voice; e. g., if a tenor talked low or a basso high. In untrained singers this situation hurts speaking as well as singing. Range is often a matter of taste. An effeminate man talks in higher voice; the overcompensating weakling may try to impress the world with a pseudobasso. Here the seed

for future voice trouble is often evident. The same happens in singing voices which are artifically changed—when the high baritone becames a tenor or the mezzo-soprano, a soprano. The result is often a divergence of registers or even their complete separation; then the high and the low range can still be functioning while the middle range disappears completely.

As to the vocal range itself, the length of cords is by no means an indicator, a fact which the voice specialist could plainly see, but not explain. Caruso, a high tenor, had very long vocal cords. Husson teaches that any correlation between vocal type and morphology of the singer is an illusion. He classifies voices according to recurrent nerve excitability and measurements of the potential range through the chronaxy of the sternocleidomastoid muscle.[4] The old classification of bassos, baritones, tenors, contraltos, mezzo-sopranos, and sopranos appears in a completely new light. The classification is now based not on the measurable length of cords or proportions between larynx and organs of the resonator, but on one factor, the excitability of the recurrent nerve, which may possibly explain all the tonal characteristics of the voice. This means, too, that the old theories on vocal registers can now be substantiated by chronaxy measurements comparing recurrent nerve and sternocleidomastoid chronaxy.[5]

5. The *cerebral origin* of tone brings us close to a mental concept. The emotional influence on tone and the tonal expression of emotional disturbances become evident. This is new physiological proof for my research on the voice of neurosis, for changing mental concepts mean a change in tonal concepts. To be sure, since the time of Scripture's research,[6] we have been close to a cerebral theory of vocal origin. He first outlined and recorded on kymograms the detailed vocal changes in neuropathology. The singing voice can be disturbed by initial symptoms which are indeed pathognomonic but which do not appear in speaking because the vocal changes are too fast. This method is called microphonic registration. For example, when a patient with multiple sclerosis sings a tone on the vowel "Ah," one finds irregularities in several kymographic waves, typical exclusively for multiple sclerosis. They are inaudible, but visible long before the classical symptom of scanning speech can be heard. Other chronic diseases of the central nervous system which reveal specific changes in singing are bulbar poliomyelitis, which occasionally affects the soft palate so slightly that nasality (open rhinolalia) cannot be heard in speaking, but in singing mild paresis is evident. This is more pronounced in myasthenia gravis, where throat, tongue, and soft palate are often initially involved. Fatigability after rest during singing is noticeable. Parkinson's disease causes a typical vocal tremor which again can be registered on a kymograph in singing long before it becomes audible. In all these central disturbances changes in the vibratory modus of vocal cords can be found.

Amusia

Amusia is a rare but interesting characteristic syndrome, which proves the cerebral origin of vocal features through their ruin in cerebrovascular accidents, which impair or destroy musical perception, musical memory, and musical performance. One can find an expressive amusia which damages the singing performance exclusively. Musical elements in singing can be lost although the speech melody is not involved. Pitch, intervals between tones, and range are affected. Often differences in intensity in singing disappear, and piano and forte singing then sound alike. The duration of tones and the pauses between tones become inexact. Occasional complete changes in range have been observed. All these phenomena could not be explained before Husson's theories.[7]

The endocrine influence on the singing voice is understandable only within the context of multiglandular interrelationships. The voice in normal or disturbed function always reflects the influence of the endocrine orchestra, of which the pituitary might be called the conductor. Not enough data are

available to draw practical conclusions on dynamic correlations between the singing voice and the endocrine system. The endocrine activity upon the voice has to be understood as involving the participation of the diencephalon. Pathological changes of voice, e. g., during changes in sex life, are not just the result of sex-hormonal influence but of general neuromuscular action. This fact will obviously have to change our viewpoints regarding hormonal therapy.

The fact that the larynx has a marked responsiveness to certain hormones may be based on the dependence of the parasympathetic, which innervates the whole endocrine system. At any rate, male hormones should never be used in female singers as long as psychogenic etiology cannot be entirely ruled out. Changes of singing during menstrual periods often occur, however uncharacteristically: hoarseness in one patient does not exclude an especially radiant voice in another. Vocal performance depends on changes in swelling of mucous membrane within the resonator, nose, oral cavity, and so on. Impedance, a sensation not unlike resonance, during singing, within the laryngobuccal canal, changes respiration and is certaintly influenced by emotional reactions of the singer to certain endocrine situations. Pregnancy changes the singing voice in 85% of women; progesterone and estradiol (Progynon) have a vasodilating effect that causes changes within cords and ventricles. The voice during pregnancy is lowered two to six notes through the increased capillary permeability. Because after gravidity the singing range returns to normal, hormonal therapy is not indicated.[8]

Conclusion

These are typical examples for a careful approach to therapy, which can be handled in the pathology of singing only with considerable experience. In general, the qualities which enable the laryngologist to become a voice specialist are such features of personality as inclination, talent, and empathy with artists. He will learn that his work can never compete with the dramatic results of surgery. The medication which he would prescribe without hesitation to the nonsinger, has to be selected most carefully; for example, epinephrine (Adrenalin), codeine, silver nitrate are "taboos" for the singer's vocal cords, and antihistamines given before a performance can be a handicap because of their drying effect.[9] In general, also, the acoustic diagnosis is at least as important as the visual one. Only by using both approaches shall we be able to understand the importance of impedance for voice training and therapy: the singer's education should involve a certain number of inner sensations repeated until eventually they can be consciously and unconsciously reproduced at any time. Sensory-motor sensations in singing will thus be fortified. Hearing will be more important than conscious breathing, which coordinates itself through the control of impedance. Only too often the vocal teacher tries to transfer his own sensations of impedance to the student, who may feel them quite differently. The singer who explains these sensations to the laryngologist is entitled to serious understanding, although he uses a different idiom.

I should like here to put in a few words about the common sources of damage to the singing voice. It should be the task of school physicians to examine hoarse children, as, for example, I have done in my role of school physician in the Richmond schools for nearly 20 years. The New York School System has an excellent health department which by its work contributes to the hygiene of voices.

Voices are easily ruined by yelling. Cheering and cheer leading should be abolished because the damage to the voice is often beyond repair.

Altogether, laryngologists, singers, and singing masters should learn to communicate more successfully with each other. The singer wants understanding and reassurance but not the punishment of voice rest applied by a physician who too frequently

confuses his interest in "Hi-Fi" with understanding of music and singing.

2000 Van Ness Ave. (9).

REFERENCES

1. Moses, P. J.: The Voice of Neurosis, New York, Grune & Stratton, Inc., 1954.

2. Arnold, G. E.: Einflüsse der Umwelt auf die Stimme, Wiss. Ztschr. Univ. Halle 3:487-490, 1956.

3. Husson, R.: The Classification of Human Voices, Bull., Nat. A. Teachers of Singing, Vol. 13, No. 4, 6-11, 1957.

4. Pommez, J.: Intérêt de la mesure de l'excitabilité neuro-musculaire en phoniatrie, Folia phoniat. 9:10-17, 1957.

5. Panconcelli-Calzia, G.: Hussons Lehre von der zerebralen Erzeugung der Stimmlippenschwingungen, Rev. laryng. (Supp.) 76:659-665, 1955.

6. Scripture, E. W.: Anwendung der graphischen Methode auf Sprache und Gesang, Leipzig, Johann Ambrosius Barth, 1927.

7. Jellinek, A.: Amusia. (On the Phenomenology and Investigation of Central Disorders of the Musical Functions), Folia phoniat. 8:124-149, 1956.

8. Imre, V.: Veränderungen der Stimme während der Schwangerschaft, Folia phoniat. 3: 224-232, 1951.

9. Moses, P. J.: Speech and Voice Therapy in Otolaryngology, Eye, Ear, Nose & Throat Month. 33:367-375, 1953.

Neurilemmoma of the Hypoglossal Nerve

Report of a Case

CLAUDE D. WINBORN, M.D.; DANIEL M. MARTINEZ, M.D.; BRUCE F. HOLDING Jr., M.D., and JAMES B. HUTCHESON, M.D., Dallas, Texas

The neurilemmoma is a benign, nonmetastasizing, well-encapsulated tumor arising from the nerve-sheath cells or Schwann cells.[1,3,28]

In the field of otolaryngology this type of tumor is most frequently associated with tumors of the acoustic nerve. However, since 1933, when Figi[10] first reported in the American literature the occurrence of this tumor in the pharynx, it is being reported with increasing frequency.

When this tumor arises in the pharynx (parapharyngeal space[24]) it is uncommon but cannot be considered a medical rarity.[13]

The exact number of such tumors that have been recorded in the international literature, has been reported variously by different authors.[2,7,8,11,12,14,16,17,20,26,27] At the present time the number approximates 50. Some of the confusion no doubt results because neurilemmomas have been reported from many various sites in the head and neck region. At times, it was difficult to ascertain from the original report the exact tumor site. Therefore, in some instances the inclusion of certain cases has been arbitrary.[13,14,16,21]

To our knowledge, pharyngeal neurilemmomas have been reported as definitely arising from the hypoglossal nerve in only two previous reports. The case to be described here will therefore make the third.

This particular tumor is known by a variety of names: neurilemmoma, neuroma, neurinoma, perineural glioma, neurilemoblastoma, fibroglioma, and its commonest synonym, the schwannoma.[1,2,28]

This myriad of names reflects the controversy that has surrounded the origin of this tumor. Boyd states that the paramount cell concerned in the formation of these tumors is the Schwann cell of the ectodermal

Submitted for publication Sept. 16, 1958.

From the Department of Otolaryngology, University of Texas, Southwestern Medical School, and Baylor University Hospital.

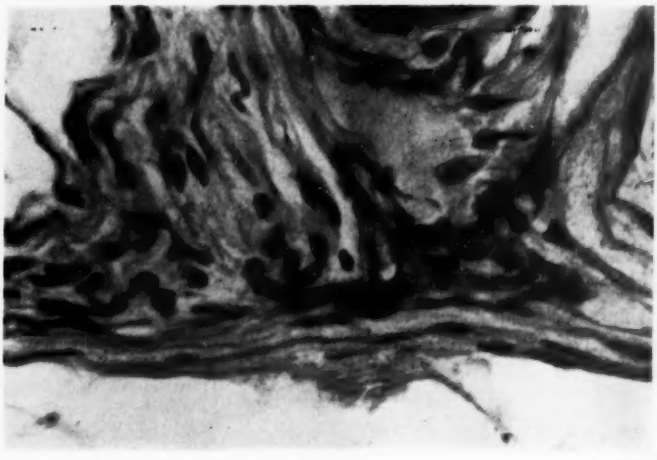


Fig. 1. — Photomicrograph showing the Schwann cells in the neurilemma of a peripheral nerve (reduced about 38% from mag. × 744).

neurilemma (Fig. 1). He adds that possibly elements of the perineurium and the endoneurium may participate in the formation of some tumors. Two patterns of microscopic architecture may usually be found in the same tumor. The more solid fascicular pattern, designated Antoni Type A, resembles a tactile corpuscle called a Verocay body. A more loosely arranged pattern of Schwann cells interspersed with cystic areas and reticulin is called Antoni Type B. The proportion of each complex varies widely from tumor to tumor.[1]

The solitary neurilemmoma should not be confused with the neurofibroma of von Recklinghausen's disease. In the latter, all the elements of the nerve are involved to some degree and nerve fibrils may be demonstrated passing through the body of the tumor, while in neurilemmoma the nerve fibrils do not seem to pass through the main bulk of the tumor. The slow-growing neurilemmoma shows little or no tendency toward malignant change, while about half of the cases of neurogenic sarcoma are found in persons with von Recklinghausen's disease.[1,23,28]

Clinical Aspects

The symptoms created by the presence of these tumors in the pharynx are directly related to size, location, and encroachment upon adjacent structures. These tumors frequently give rise to such signs as hoarseness, dysphagia, and a Horner's syndrome,[2,4] the last being indicative of cervical sympathetic involvement, while hoarseness and dysphagia may reflect a vagus nerve lesion. Objective evidence of 10th nerve involvement may at times be present as a vocal cord paralysis or paresis of the palatal and pharyngeal musculature. Dysphagia may simply represent mechanical obstruction due to the size and location of the tumor mass. The presence of the tumor mass, without other complaints, may be the presenting symptom. It is usually the intraoral mass that it first noticed, although at times cervical enlargement may be the first observed. Occasionally the patient may be completely unaware of the tumor and its occurrence is found during routine examination.

The cervical sympathetic and vagus appear to be the nerves of predilection for the pharyngeal neurilemmoma. Thus, palsies of the 10th nerve and Horner's syndrome are the two commonest neurological findings.[4,7,8,17] Apparently any of the nerve trunks are susceptible to this tumor, and in many instances the exact nerve of origin is not discernible. In the previously reported cases of pharyngeal neurilemmoma arising from the hypoglossal nerve, neither had demonstrable injurious effect on the nerve preoperatively.

On physical examination, a neurilemmoma presents as a fairly well-delineated, ovoid mass in the lateral pharyngeal area.[3] The overlying mucosa appears relatively normal and is not fixed to the tumor. The usual site of appearance is just posterior to the posterior tonsillar pillar on the lateral wall of the pharynx. The degree of distortion of the adjacent structures and the extent of encroachment on the air and food passages vary with the size of the tumor. The mass itself is usually firm but not hard. At times it may feel almost cystic in consistency. This is probably proportional to the extent of hemorrhagic necrosis or Antoni Type B tissue comprising the tumor.

Slaughter and dePeyster have described four features that would typify the presence of a pharyngeal neurilemmoma of cranial nerve origin.[13] These are the following: a submucosal tumor presenting in the posterolateral pharyngeal area; the history of slow growth; medial displacement of the internal carotid artery as determined by palpation, and evidence of paresis of the cranial nerve involved. Admittedly, however, all of these features are by no means constant.

Report of Case

A 35-year-old white housewife presented herself with the complaint of a mass in the throat and difficulty in swallowing for six weeks, although she had noted swelling in the neck for about 18 months.

Her general physical examination was not remarkable, and her neurological examination was completely normal. The pertinent E. N. T. findings were as follows: In the left lateral pharyngeal area was an ovoid, firm tumor that displaced the tonsillar pillars anteriorly and was covered by normal mucosa. The soft palate was displaced anteriorly and medialward. The mass occupied about one-half of the aperture of the faucial isthmus. The tumor extended from the base of the skull to the tip of the epiglottis. In the neck a firm mass was palpable along the anterior border of the sternomastoid muscle and extended from the greater cornu of the hyoid bone to the mastoid process. The mass was semifixed, but the skin was freely movable. The tip of the parotid gland also felt slightly nodular and prominent.

A biopsy specimen was then taken under local anesthesia. To accomplish this the mucous membrane and pharyngeal musculature were incised longitudinally under direct vision. A large cup forceps was used to take adequate tissue for microscopic examination. The small incision was sutured. The biopsy was compatible with neurilemmoma. At surgery, four days later, the biopsy site had healed to the point of closure without infection.

Operation.—Under local anesthesia, through a small transverse incision, a low tracheostomy was performed. Through this, endotracheal general anesthesia was then administered. A submandibular incision was made from the greater cornu of the hyoid bone curving upward below the angle of the mandible along the anterior border of the sternomastoid muscle to the mastoid tip. The skin flaps were developed, which included the platysma muscle. The marginal mandibular branch of the facial nerve was identified and preserved. The submaxillary salivary gland was dissected free from its bed and retracted anteriorly after the facial vessels had been ligated. The tip of the parotid gland was resected, as it was edematous and compressed lateralward by the tumor. The stylohyoid muscle was separated from the hyoid, thus allowing this muscle and the diagastric muscle to be retracted superiorly. The sternomastoid was retracted posteriorly. The tumor could then be visualized as a well-encapsulated tubular-shaped mass extending from the posterior border of the greater cornu of the hyoid bone to disappear behind the ascending ramus of the mandible and the mastoid tip. The occipital artery was compressed anteriorly and had to be ligated. The contents of the carotid sheath were then identified and preserved. The tumor could be palpated extending to the base of the skull and was separated in this area by blunt finger dissection. It was then discernible that the tumor completely encompassed the hypoglossal nerve and appeared to be arising directly from it. The tumor could be separated from the hypoglossal nerve by a stripping type action until the descending branch of the hypoglossal was encountered. In this area further stripping of the tumor was impossible; therefore, the nerve was sectioned. The remaining portion of the tumor was then dissected with relative ease. The vagus, sympathetic, glossopharyngeal, and spinal accessory nerves were all identified and appeared to be free of involvement. The hypoglossal nerve was anastomosed end-to-end and the wound closed. A nasogastric feeding tube was inserted. There was no excessive blood loss or other surgical complications.

Immediately after operation the patient had a left hypoglossal nerve paralysis together with a paralyzed left vocal cord. This resulted in hoarseness and dysphagia. The feeding tube was left in place for seven days and the tracheostomy maintained for two weeks. At the time of this writing, some six months after surgery, the hoarseness and dysphagia have cleared, and the hypoglossal nerve shows evidence of recovery. It was felt that damage to the vagus nerve was incurred during the dissection of the tumor from the base of the skull, as this was the only point at which the vagus was not under direct visualization. The tracheostomy and submandibular incisions have healed in a fine line scar. It should be mentioned that the tumor was compressed into a tubular-shaped mass in the neck, but after excision it assumed a more ovoid form (Figs. 2 and 3).

Comments and Conclusions

The question has been previously posed as to whether such tumors should be surgically removed or left in situ.[8,18] Certainly if these tumors are to be actively treated at all, then surgical excision is the

Fig. 2.—External and sectioned surfaces of tumor, which measured $6 \times 5 \times 3.2$ cm. and was completely encompassed by a fibrous capsule. Its interior was moderately firm, solid, pinkish-tan and mottled by recent hemorrhages. The tumor assumed an ovoid shape after excision but in situ appeared compressed into a more tubular form.

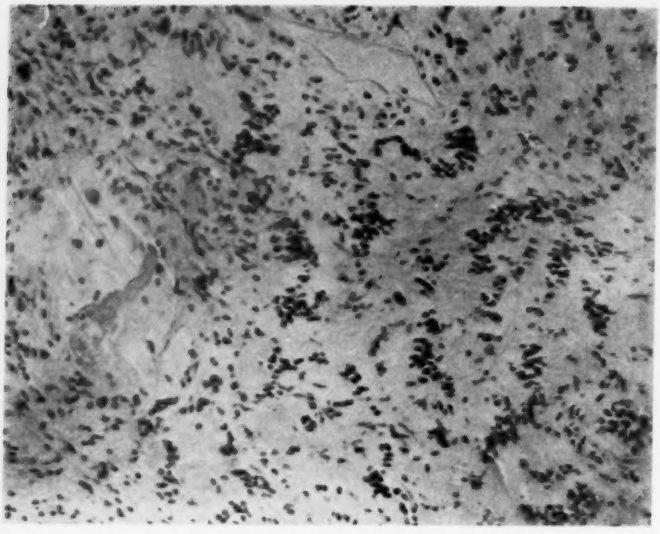


Fig. 3. — Photomicrograph of tumor showing the palisaded nuclei with focus of cystic degeneration (reduced about 8% from mag. × 100).

only method. It is well established that the neurilemmoma is extremely radioresistant, and irradiation to the area only adds to the problem of surgical attack at a later date.[8,19,20-23] The decision as to whether these tumors should be removed is dependent upon many factors, and each case is entitled to a complete evaluation of its own peculiarities. The prime considerations are the following:

1. The tumor is very slow growing and rarely if ever becomes malignant.[1,2,5,9,19,23]
2. The size and location of the tumor
3. The encroachment of the tumor upon contiguous structures
4. General mental and physical condition of the patient
5. The anticipated surgical results versus the operative morbidity.

Because of the location of these pharyngeal tumors, with their adjacent nerve and vascular relations, they are considered by some to be malignant by position.[13,25] It is a reasonable assumption that the symptoms and disabilities of the patient will progress as the tumor continues to enlarge and that the optimum time for excision is when the tumor is first discovered. Ranta, on the other hand, has followed a patient with a neurilemmoma for six years with no apparent ill effects other than a slight increase in size.[7] When major vessels and nerves are intimately related to such a tumor, and when these structures often have to be excised in order to remove the tumor, the operative morbidity may far outweigh the relief of the initial symptoms. In some instances the tumor may be removed without sacrificing the involved nerve, but cranial nerve palsies account for the large majority of cases of postoperative morbidity.[23]

In the case presented here, we felt that the patient's symptoms were sufficient to warrant surgical procedure. In addition, her general physical condition was excellent, and her mental processes were of such a nature that the presence of the mass alone was very disconcerting.

There appears to be much controversy over the best avenue of surgical approach and the relative merits of tracheostomy. Neurilemmomas have been successfully and adequately removed both via the intraoral approach and via the external approach through the neck.[3,5,6,8,9,12,13,23] They have been removed with and without tracheostomy. Once again the correct answer would seem to be dictated by the individual case.

It appears that the intraoral approach would be limited to relatively small, extremely well-demarcated tumors. The po-

tential hazards of such an approach would be more pronounced too; for it is a well-experienced fact that any such mass is usually more extensive than is appreciated by palpation. Adequate surgical exposure through the intraoral approach would appear as a major obstacle. The visualization and thus preservation of major vessels and nerves would be limited.[8] With inadequate exposure even a relatively small amount of obscuring blood flow presents a formidable barrier; and in the event of major hemorrhage, the result may be catastrophic. The chief advantage of the intraoral approach would seem to be lack of an external scar.

The external approach, on the other hand, seems more widely applicable regardless of the size of the tumor. The advantages of adequate exposure with identification and protection of important structures are at once obvious. Through the external approach the arterial supply to the area is under direct vision and therefore under control at all times. If the skin creases are utilized to the fullest advantage, the external scarring can be reduced to a minimum.

Whether or not tracheostomy is performed in such cases is again dependent on several factors, but seems less of a decision than some of the others. If the tumor is of such small size and the anticipated surgical manipulation of such minor endeavor that the intraoral route is practicable, then tracheostomy would hardly seem indicated.

If there is any procedure that can be considered as routine in our head and neck surgery, then it is tracheostomy. True, we do not use tracheostomy in all head and neck cases, but it is used in a vast majority of them. Whenever there is the slightest question as to the need of tracheostomy, then it is performed without hesitation. Besides the more frequently emphasized indications for tracheostomy, such as preservation of an airway and the maintenance of adequate tracheobronchial toilet, there are several important ones less frequently discussed. Whenever there is chance of injury to the vagus nerve or its laryngeal branches, tracheostomy should be performed.[23]

The preoperative diagnosis of neurilemmoma can only be ascertained with certainty by familiarity with the characteristics of pharyngeal tumors, a high index of suspicion, and above all, by adequate tissue biopsy.

Needle biopsy, in some hands, appears to be the method of choice in this type of tumor. In our experience, direct visualization with biopsy yields a higher percentage of reliable data. If needle biopsy provides enough representative tissue for a positive diagnosis, then all is well. Frequently, inconclusive results are obtained, and direct tissue biopsy must then be done. We do not feel that incision of the pharyngeal mucosa and musculature is an increased hazard, nor do we feel the probability of contamination of the pharyngeal space is a very likely one. If a few days elapse between the transmucosal biopsy and the definitive neck surgery, the mucosal incision will be sufficiently sealed to prevent complications.

Summary

Neurilemmoma is a benign tumor of nerve-sheath origin that occasionally arises from the cranial nerves as they traverse the neck.

There is a brief presentation concerning the origin, clinical characteristics, and behavior of these tumors.

There are two previous cases recorded where a pharyngeal neurilemmoma arose from the hypoglossal nerve, and the third such case is presented.

A discussion of the indications for operation, the choice of incision, the benefits of tracheostomy and direct tissue biopsy are included.

3707 Gaston Ave.

REFERENCES

1. Boyd, W.: Pathology for the Surgeon. Ed. 7, Philadelphia, W. B. Saunders Company, 1955, Chap. 27.

2. Sommer, K.: Neurilemmoma of the Pharynx, Ann. Otol. Rhin. & Laryng. 61:636, 1952.

3. Violé, P.: Schwannoma of the Pharynx, Ann. Otol. Rhin. & Laryng. 55:334, 1946.

4. Manning, E. L.: Solitary Neurofibroma Presenting in Pharynx and Neck with Horner's Syndrome, A. M. A. Arch. Otolaryng. 58:740, 1953.

5. Macial-Rojas, R. A., and Picó, J.: Neurilemmoma of the Pharynx, A. M. A. Arch. Otolaryng. 63:93, 1956.

6. Hanley, J. S., and Davol, R. T.: Pharyngeal Neurilemmoma, A. M. A. Arch. Otolaryng. 56:207, 1952.

7. Ranta, L.: Neurogenous Tumors of the Pharynx, Acta oto-laryng. 45:153, 1955.

8. Guggenheim, P.: Schwannoma of the Pharynx, J. Internat. Coll. Surgeons 19:450, 1953.

9. Kennon, W. G., Jr.: Neurinoma of the Pharynx, Laryngoscope 60:388, 1950.

10. Figi, F. A.: Solitary Neurofibroma of the Pharynx, A. M. A. Arch. Otolaryng. 17:386, 1933.

11. Varvin, P.: A Case of Solitary Neurogenic Tumor in the Pharynx, Acta oto-laryng. 39:256, 1951.

12. Kirchner, J. A., and Sheld, D. P.: Schwannoma of the Pharynx, Connecticut M. J. 20:284, 1956.

13. Slaughter, D. P., and dePeyster, F. A.: Pharyngeal Neurilemmomas of Cranial Nerve Origin, Arch Surg. 59:386, 1949.

14. Stout, A. P.: Peripheral Manifestations of Specific Nerve Sheath Tumor (Neurilemmoma), Am. J. Cancer 24:751, 1935.

15. Friedman, L., and Eisenberg, A. A.: Neurofibroma of the Hypoglossal Nerve, Ann. Surg. 101: 834, 1935.

16. Møller, E.: 2 Tilfaelde of Solitaere Neurinomer af Acusticus-Typen det ene lokaliseret til Spatium parapharyngeum det andet til Glandula thyreoidea, Nord. Med. 16:3175, 1942.

17. Saxén, E.: Tumours of the Sheaths of the Periphereal Nerves, Acta path. et microbiol. scandinav., Supp. 89, 1948.

18. Wolf, G. D., and Gilson, S.: Neurilemmoma (Schwannoma of the Neck), New York J. Med., 54:2484, 1954.

19. Ehrlich, H. E., and Martin, H.: Schwannomas (Neurilemmomas) in the Head and Neck, Surg. Gynec. & Obst. 76:577, 1943.

20. Koop, C. E.; Jordan, H. E., and Horn, R. C., Jr.: Neurilemmoma of the Pharynx, Surg. Gynec. & Obst. 85:641, 1947.

21. Cutler, E. C., and Gross, R. E.: Neurofibroma and Neurofibrosarcoma of Peripheral Nerves: Unassociated with von Recklinghausen's Disease, Arch. Surg. 33:733, 1936.

22. Johnson, F., and Fisher, R.: Schwannoma of the Pharynx with Horner's Syndrome, Laryngoscope 67:815, 1957.

23. Conley, J. J.: Neurogenous Tumors in the Neck, A. M. A. Arch. Otolaryng. 61:167, 1955.

24. Cranner, L.: Neurofibroma of the Parapharyngeal Space, Ann. Otol. Rhin. & Laryng. 67: 178, 1958.

25. Motley, F. E.: Position Malignancy, Arch. Otolaryng. 34:771, 1941.

26. Lemariey, Muler, and Lande: Un cas de tumeur para-pharyngée (schwannome), Ann. otolaryng. 70:50, 1953.

27. Gekker: Two Observations on Neuroma of the Pharynx, Vestnik Oto-Rino-Laring., 16:2, 1954.

28. Stout, A. P.: Tumors of the Peripheral Nervous System, in Atlas of Tumor Pathology, Armed Forces Institute of Pathology, 1949, Sec. 11, Fasc. 6.

Laryngofissure: Indications and Technique

JOHN C. LILLIE, M.D., and KENNETH D. DEVINE, M.D., Rochester, Minn.

Laryngofissure is defined in Dorland's dictionary [1] as follows: "The operation of opening the larynx by a median incision through the thyroid cartilage with the formation of a wide window: for the removal of cancer of the larynx; median laryngotomy." This operation is an extremely useful and practical tool in the practice of the laryngologist. However, its applicability has certain limitations; if these are disregarded, its use may be hazardous to the continued well-being of the patient.

Indications

At the Mayo Clinic, we attempt to confine the operation to a few situations. In the treatment of carcinoma of the larynx, we consider that if the lesion is confined to one vocal cord, if it does not extend across the anterior commissure or posterior to the vocal process, and if the vocal cord is not fixed, then laryngofissure may be expected to result in the percentages of five-year cures reported by Schall,[2] LeJeune and Lynch,[3] Jackson,[4] and Clerf.[5] These figures range from 75% to 92%. By the same token, as emphasized by Putney and Vicens,[6] any attempt to overstep these same indications will reduce the percentage of cures, increase the morbidity and mortality rates, and defeat the principle that the first attack on cancer should be an "all out" effort. Needless to say, the malignancy of the lesion must have been proved by biopsy and histologic diagnosis.

Laryngofissure also is utilized properly in the treatment of benign conditions of the larynx that are not amenable to treatment by transoral procedures; these conditions include certain ventricular cysts, posttraumatic stenosis, extensive papillomatosis, and leukoplakia.[7] It is also done in conjunction with arytenoidectomy [8] for bilateral fixed vocal cords. Another indication for laryngofissure is that relatively rare instance in which the lesion is not adequately visible by either indirect or direct laryngoscopy or in which any doubt exists about the condition present within the larynx.

Surgical Technique

In the prebiopsy discussion with the patient, we usually go over the procedure to be carried out if the results of biopsy are positive. Briefly, this consists of one period of anesthesia during which suspension laryngoscopy is performed and biopsy done. An intratracheal tube is then placed through the mouth, and the suspension apparatus is removed. The patient is prepared, draped, and tracheotomy is done, after which the intratracheal tube is removed through the mouth, and a cuffed tube is introduced through the tracheal stoma. Laryngofissure then is performed. This combined procedure saves the patient a few days of hospitalization and avoids a second anesthetic.

After the malignancy of the lesion is established at biopsy, as just indicated, the patient is placed on the operating table so that the shoulders are slightly elevated and the head and neck are somewhat hyperextended. The anesthesiologist inserts a needle in a vein of the arm for the intravenous administration of thiopental (Pentothal) so-

Submitted for publication Aug. 8, 1958.

Read before the Section on Laryngology, Otology, and Rhinology at the 107th Annual Meeting of the American Medical Association, San Francisco, June 26, 1958.

Section of Plastic Surgery, Mayo Clinic and Mayo Foundation. The Mayo Foundation, Rochester, Minn., is a part of the Graduate School of the University of Minnesota.

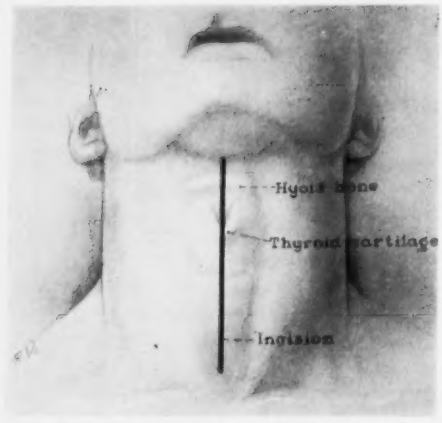


Fig. 1.—Landmarks and site of incision.

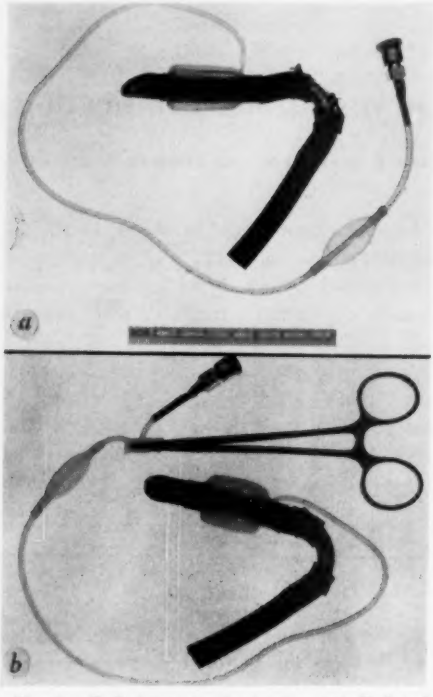


Fig. 2.—Cuffed intratracheal tube. (*a*) Deflated. (*b*) Inflated.

dium and also fluids. The skin of the neck is prepared by cleansing with soap and water, and then with alcohol; tincture of thimerosal (Merthiolate) then is applied. Drapes are placed, and the midline of the neck from the thyroid cartilage to the suprasternal notch is infiltrated with a 1% solution of procaine containing 0.25 ml. (4 minims) of a 1:1,000 solution of epinephrine per 30 ml. A midline incision in the neck is made (Fig. 1), and tracheotomy is done below the thyroid isthmus; 0.5 ml. of a 10% solution of cocaine is injected into the trachea before it is opened. The cuffed intratracheal tube is then introduced; the cuff is inflated, and the tube is connected to the gas machine (Fig. 2). At this point, nothing further is done until the patient is put to sleep by the intravenous injection of thiopental sodium and inhalation of a nitrous oxide-oxygen mixture. The presence of a tight connection with the gas machine also is demonstrated; this permits positive pressure to be used if necessary.

The incision is now extended in the midline to the hyoid bone, cutting through the perichondrium of the thyroid cartilage only in the midline (Fig. 3*a*). Hooks are placed on each wing of the thyroid cartilage at its notch, and firm but gentle lateral traction is applied. If the cartilage is soft, as in some women, the thyroid cartilage may be cut through with a scalpel; otherwise, an oscillating saw is used, care being taken to avoid cutting through the internal perichondrium (Fig. 3*b*). Two saw cuts occasionally are made, leaving a strip of thyroid cartilage attached to the specimen that can be utilized as a "handle." This step affords wider clearance at the anterior commissure. The larynx is entered with the scalpel by making an incision in the cricothyroid membrane and then in the midline of the internal perichondrium of the thyroid cartilage and mucous membrane of the larynx. Further traction exposes the interior of the larynx, the mucous membrane of which is painted with a 10% solution of cocaine on cotton applicators. The tumor is now inspected at first hand (Fig. 3*c*).

A step that we have found often reduces bleeding is then carried out; this consists of injection of the local-anesthetic solution along the internal perichondrium of the involved side, which causes the solution to balloon up beneath the true cord, ventricle,

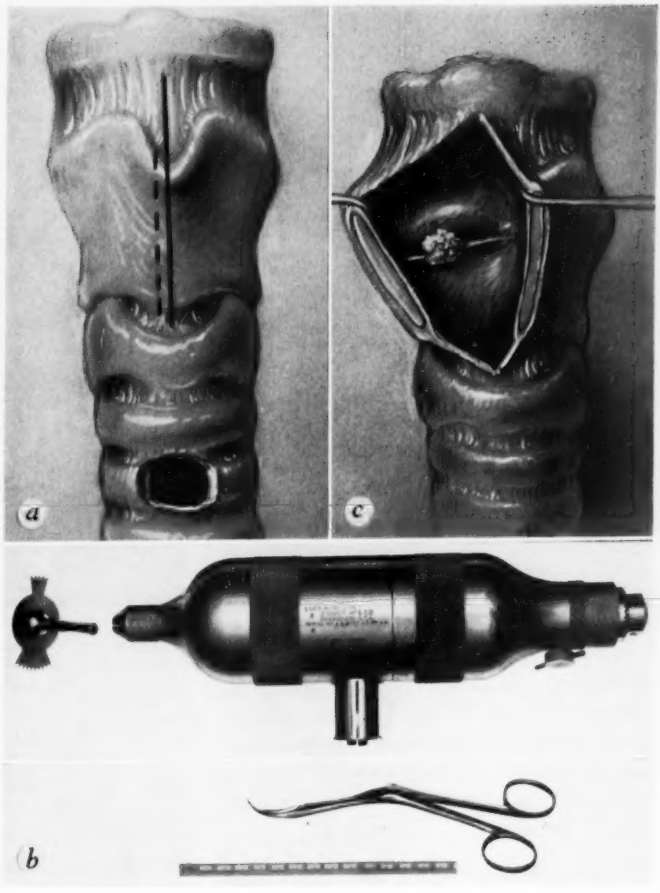


Fig. 3.—(*a*) Tracheotomy opening. Solid line shows usual incision of thyroid cartilage; dotted line shows additional incision when "handle" of cartilage is used. (*b*) Motor-driven saw and right-angled scissors. (*c*) Exposure obtained, showing growth.

and false cord. Vertical incisions then are made below the true cord and above the false cord, carrying them to the cartilage. The resulting wedge of tissue is grasped at the midline with forceps, and dissection is carried out along the cartilage to remove the mass en bloc (Fig. 4). If a bleeding vessel is encountered during this stage, it is clamped, ligation being done later. The block of tissue containing the tumor is removed, after the tissue is freed down to the vocal process, by cutting with a right-angled scissors (Fig. 5). The raw bed is packed temporarily with gauze while the specimen is examined and then sent immediately to the pathologist, who determines by the frozen-section technique whether the borders of the removed tissue are free of tumor.

Bleeding vessels are ligated, and the capillary oozing is controlled by electrocoagulation, care being taken to attain thorough hemostasis of the bed. The wound is closed with interrupted absorbable surgical sutures, approximating the fascia in the midline. Sutures are not placed in cartilage or perichondrium. The cutaneous edges are approximated with interrupted sutures of silk, beginning at the upper end of the incision. When the tracheal stoma is approached, the cuffed intratracheal tube may be deflated and removed, and a No. 7 tracheotomy tube put in its place. The few sutures remaining are put in place before the patient awakes.

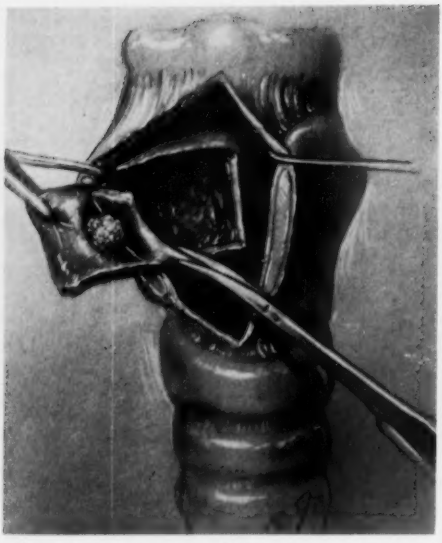


Fig. 4.—Removal of false cord, ventricle, true cord, and subglottic mucous membrane en bloc.

care being taken not to close tightly around the tracheotomy tube.

The final step is the application of a snug dry dressing over the incision above the tracheotomy tube. By the time the dressing is applied, the patient is usually awake; if not, he is sent to the postoperative recovery room until he is awake. During this time, care of the airway is maintained.

Postoperative Care

When the patient returns to his room, he is awake. The room has been prepared previously with a steamer, a croup tent, and an electrically driven suction apparatus. We occasionally use the "Mist O_2 Gen" apparatus hooked over the tracheotomy tube if the secretions tend to become thick. One of the most important phases of postoperative care is that of the tracheotomy tube. Secretions are aspirated every hour routinely or more often if necessary for the first 24 hours. The inner tube is cleaned at least every four hours.

Patients are encouraged to get out of bed the evening of the operation and are allowed up at will after that. Sips of fluids are given the first postoperative day; if they are not tolerated readily, the patients often

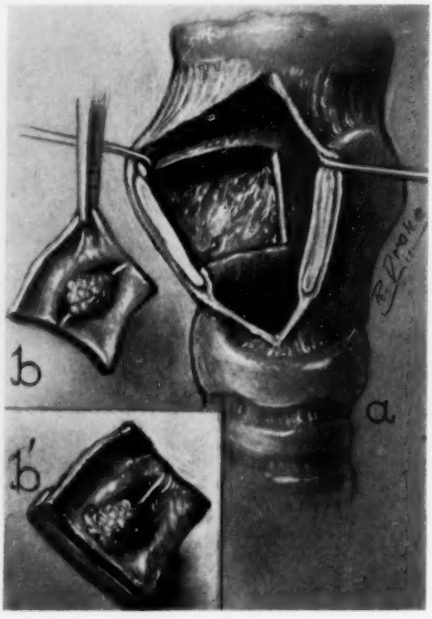


Fig. 5.—(*a*) Defect remaining after removal of specimen. (*b*) Removed specimen. (*b'*) Specimen with "handle" of cartilage included.

can handle a diet of semisolid foods such as Jello, custards, and mashed potatoes. A feeding tube is used in about half of the cases. Most patients are able to swallow without choking in one or two days; until then, their fluid intake can be maintained by the intravenous injection of solutions with or without the addition of one of the soluble vitamin preparations.

During the postoperative stay in the hospital, which usually averages from five to seven days, a suitable antibiotic is given daily. The tracheotomy tube frequently can be removed in two to four days, as determined by the patient's ability to breathe adequately with the tube corked or by indirect laryngoscopy to note if the size of the airway is adequate. It is not necessary to close the tracheal stoma surgically, since that occurs spontaneously in one or two days. All that is needed is a dry dressing, which is changed as often as needed as it becomes wet with mucus. Sutures are removed in approximately seven days. We do not discourage use of the voice but do not

encourage its employment in more than ordinary conversation.

Comment

In the five-year period from 1952 to 1956, inclusive, there were 238 laryngofissures performed at the clinic as compared with 282 laryngectomies. If the diagnosis could be made earlier, it would be anticipated that these numbers would be reversed.

The technique of performing laryngofissure as we have done it in the past several years does not differ greatly from methods used elsewhere except in two steps. As already noted, we prefer to do the preliminary tracheotomy with the patient under local anesthesia, removing a button of a tracheal ring, usually below the thyroid isthmus, and then to insert the cuffed intratracheal tube and carry out the rest of the procedure using general anesthesia. There are several reasons for this. Not all patients are of a suitable psychic make-up to undergo with equanimity this or any other type of operation while they are awake. We are better able to control the airway and also are able to prevent aspiration of blood and mucus into the trachea, with its resulting coughing, during the operative procedure. These preliminary steps also prevent, insofar as possible, postoperative pulmonary complications. They also reduce trauma to the surgeon's nervous system because (1) the patient is safely quiet; (2) we have a means by which blood and secretions are properly and promptly evacuated in the postoperative period, and (3) we know that if bleeding occurs in the first 24 hours we are much less likely to encounter serious trouble while the bleeding is being controlled.

The procedure that we have described is used in an ideal case. Because carcinoma of the larynx is a vagarious disease, variations in the technique used for its treatment occasionally are required. Thus, it may be necessary only to excise the true cord in an early cordal lesion. Another variation would be excision of one cord and part of another. The mucous membrane of the uninvolved cord occasionally may present so-called smoker's epithelium. It is expedient to remove the affected mucous membrane from such a cord and close the edges of healthy mucous membrane with fine absorbable surgical sutures after excising the opposite cord that contained the carcinoma.

Before being dismissed from our care, patients are told that routine periodic checkups are vital to their well-being. They are reminded of the possibility that granulation tissue may form and that the voice always may be somewhat weak, two facts that are presented to all patients in the preoperative discussion, along with a description of the operation and why it is being recommended. We have found that giving all patients a mimeographed summary of the preoperative and postoperative discussion has been highly satisfactory with regard to their understanding and peace of mind.

Mayo Clinic.

REFERENCES

1. Dorland's Illustrated Medical Dictionary, Ed. 23, Philadelphia, W. B. Saunders Company, 1957, p. 730.

2. Schall, L. A.: Carcinoma of the Larynx, New England J. Med. 229:574-576 (Oct. 7) 1943.

3. LeJeune, F. E., and Lynch, M. G.: Value of Laryngofissure Operation, Ann. Otol. Rhin. & Laryng. 64:256-262 (March) 1955.

4. Jackson, C. L.: Laryngofissure for Cancer of Larynx: Observations Based on Series of 50 Consecutive Cases, Arch. Otolaryng. 33:520-535 (April) 1941.

5. Clerf, L. H.: Cancer of the Larynx: Analysis of 250 Operative Cases, Arch. Otolaryng. 32:484-498 (Sept.) 1940.

6. Putney, F. J., and Vicens, E. A.: Surgical Treatment of Carcinoma of the Anterior Commissure of the Larynx, Laryngoscope 66:1041-1048 (Aug.) 1956.

7. Cracovaner, A. J., and Rubenstein, A. S.: The Management of Laryngeal Keratosis by Laryngofissure, A. M. A. Arch. Otolaryng. 63: 203-207 (Feb.) 1956.

8. DeBord, B.: Surgical Management of Laryngeal Disorders by the Laryngofissure Approach, Laryngoscope 67:360-364 (April) 1957.

Pharyngeal-Esophageal Diverticulum

JOSÉ A. ARRUDA BOTELHO, M.D., Sao Paulo, Brazil

The most adequate process to cure the pharyngeal-esophageal diverticulum seems to be its surgical removal. In Brazil, we must emphasize the work of E. Vasconcelos, who studied the subject very thoroughly. Nowadays, the surgical removal of the diverticulum can be done safely, owing to antibiotics. The era when one small opening in the neck of the diverticulum represented an almost certain mediastinitis, with great risk to the patient's life, is now over. Thus the surgical removal of the pharyngeal-esophageal diverticulum has been done by many outstanding surgeons in all countries. Sometimes it is not easy to reach the diverticulum which extends into the chest, especially in patients with short necks. In order to make the surgery easier we suggest the following technique:

1. Before the patient goes under general anesthesia we locally anesthetize one of the nostrils in order to pass a Nelaton catheter (No. 12 or 14) through it. In one end of the catheter we fix a small rubber balloon, made up by inserting a small metallic or plastic tube no longer than 5 mm. inside the end. In this way it is possible to tie around a piece of Penrose drain already tied up on the other end. By using a syringe it is possible to inflate the small rubber balloon through the Nelaton catheter. In order to make the introduction of this catheter easier we usually put inside it a guitar string. With the patient seated with his head bent forward, we introduce the catheter through the nostril previously anesthetized. We shall notice the catheter going down easily until it reaches the diverticulum sac, where it stops. We tie the catheter with a tape around the patient's nose and take out the guitar string so we can inflate the balloon and look at its bump on the neck. This enables us to make our decision, whether we are going to operate on the right or the left side of the patient.

It is possible to wait until the patient is asleep to introduce the balloon. In a patient with a large diverticulum it is even better to do it this way because the balloon goes into the diverticulum easily and the patient does not feel anything. In a patient with a small diverticulum and mild dysphagia, it may be difficult to make the ballooned catheter go into the diverticulum, so it is advisable to introduce the catheter into the diverticulum with the patient seated with his head bent forward.

Submitted for publication July 21, 1958.

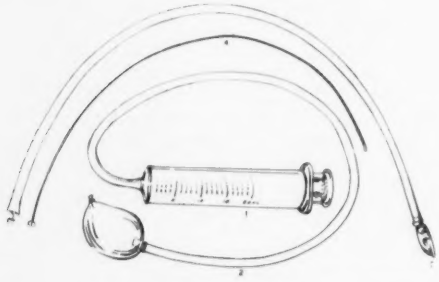
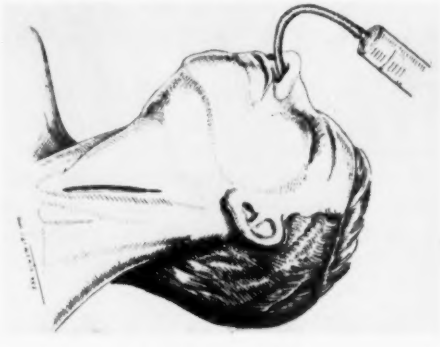


Fig. 1.—(1) Syringe. (2) Ballooned catheter. (3) Balloon made of a piece of Penrose drain. (4) Guitar string.

Fig. 2.—Ballooned catheter in place.



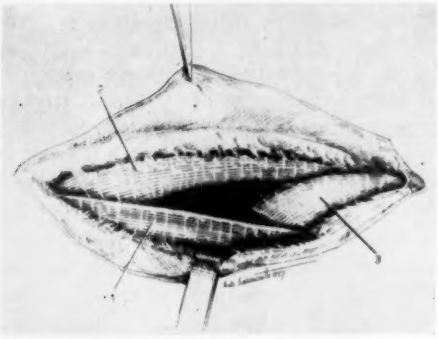


Fig. 3.—(1) Sternocleidomastoid muscle. (2) Sternohyoid muscle. (3) Omohyoid muscle.

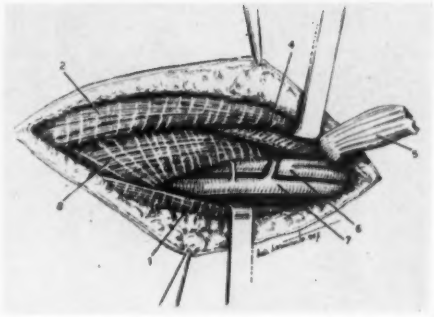


Fig. 4.—(4) Thyrohyoid muscle. (5) Omohyoid muscle sectioned. (6) Carotid artery. (7) Jugular vein. (8) Sternothyroid muscle.

2. We start the operation with an incision following the anterior edge of the sternocleidomastoid muscle in its inferior two-thirds.

3. After making the skin hemostasis, we cut the cervical superficial fascia, following the anterior edge of the sternocleidomastoid muscle and with holders we push the muscle laterally.

4. After pushing the sternocleidomastoid muscle laterally we can see, crossing the surgical field from up to down and from right to left, the omohyoid muscle. We cut this muscle and the fascia cervicalis media.

5. Now we can see the large vessels of the neck, which should be kept aside by the holders. To do that it is necessary to tie and cut the thyrolinguofacial trunk and the middle thyroid vein.

6. So far in the operation the larynx, the pharynx, and the cervical esophagus are still covered by the thyroid lobe and also by the infrahyoid muscles, especially by the sternothyroid. We must cut this muscle close to its insertion in the thyroid cartilage.

7. Now we can see the lobe of the thyroid gland, and it is easy to tie and cut the superior thyroid artery.

8. The lobe of the thyroid gland can be tilted by pushing its superior pole forward and downward. This shows the thyroid cartilage with the insertion of the inferior constrictor of the pharynx on the oblique line.

9. With a light holder inserted behind the posterior edge of the thyroid cartilage, we pull it forward, making it at the same time turn to the opposite side. Thus we can show a part of the pharyngeal posterior wall.

Fig. 5.—(9) Superior carotid artery. (10) Thyroid gland lobe tilted forward and downward. (12) Diverticulum sac inflated by the balloon. (13) Inferior pharyngeal constrictor muscle.

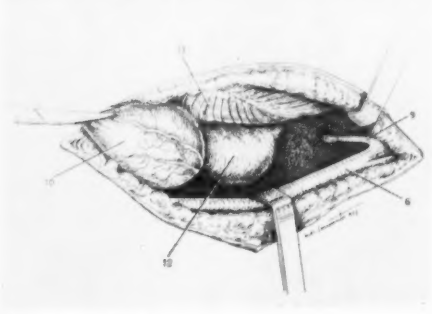
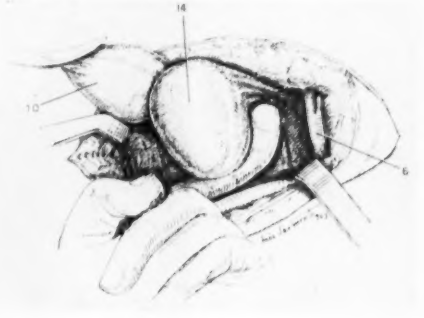


Fig. 6.—(14) Free diverticulum sac.



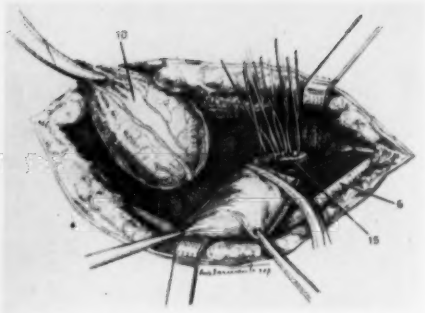


Fig. 7.—(15) Section of the neck of the diverticulum and suture of the pharyngeal wall.

the prevertebral space, and anterior part of the vertebral body.

10. Now the dissection reaches the place where the diverticulum is supposed to be. It can be found with great accuracy if we ask the anesthetist to inflate with 15 or 20 cc. of air the small balloon that should be inside it. The great advantage of the method here suggested is that we can locate the diverticulum without any trouble. Besides the inflated rubber balloon inside it gives it a round shape and some elastic rigidity, allowing us to circumscribe it easily with the index finger and to bring it inside the operative field.

11. When the diverticulum is completely free from adhesions and we can see its neck, we should ask the anesthetist to empty the ballon and withdraw the ballooned catheter.

12. Before starting to suture the neck of the diverticulum it is advisable to introduce a thick catheter through the esophagus. We usually ask the anesthetist to introduce in the patient's esophagus one of the largest tracheal catheters. When this is done, there will be no danger that the suture will produce a stricture in the mouth of the esophagus. To suture the neck of the diverticulum we grasp it with a forceps and start cutting it bit by bit so we can put a stitch after each small cut, making a kind of Sweet suture.

13. After we take out the diverticulum the thyroid gland lobe gets back to its place, and we tie it with a stitch in its pole.

14. A Penrose drain is placed in the wound, and stitches tie together the superficial cervical fascia. The skin is sutured, leaving one end of the Penrose drain out.

15. We feed the patient through a nasal catheter during the first week after the operation.

Comments

We call the attention to the fact that we get close to the pharynx by tilting the lobe of the thyroid gland. We push its pole forward and downward instead of going to the inferior pole, which would increase the danger of injuring the recurrent nerve.

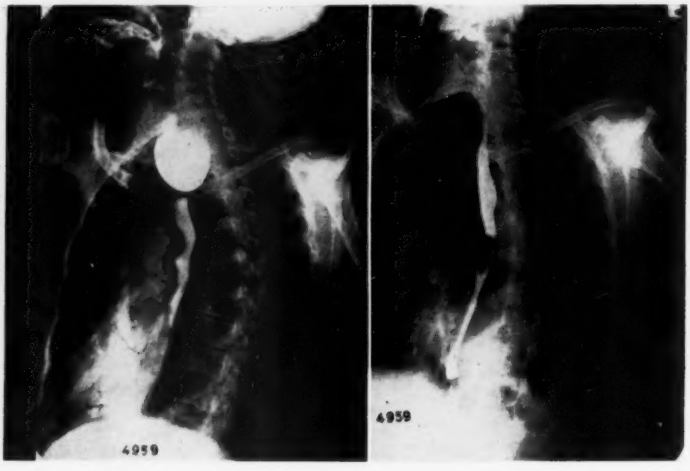


Fig. 8. — Example of one of six cases—before and after operation—in which the technique described in the text was used.

Using this technique we have operated in six cases already. In the first case the patient had stricture in the mouth of the esophagus because we did not use the thick catheter in the esophagus before suturing the diverticulum neck. This patient has been subjected to esophageal dilatations and is now cured.

In the other five cases the patients had uneventful recoveries. In all cases we made a small incision in the cricopharyngeal muscle after taking out the diverticulum, as is advised by many outstanding surgeons, since it may help to prevent recurrence of the diverticulum.

Rua Beneficiência Portuguesa 9.

A Comparative Study of Nasal Decongestion by Sympathomimetic Drugs

DOMINGO M. AVIADO Jr., M.D., Philadelphia; A. L. WNUCK, B.S., and EDWIN S. DE BEER, Ph.D., Tuckahoe, N. Y.

The current widespread use of about 15 sympathomimetic drugs to decongest the nasal mucosa has provoked the question of their comparative effects. There are two approaches in obtaining direct evidence for nasal decongestion. In man, there are several methods that have been introduced to measure nasal resistance (see references cited in monograph by Uddstromer,[1] 1940). Unfortunately, only ephedrine[2] and methamphetamine[3] have been studied with the benefit of such methods. In 1939, Jackson[4] introduced a method using dogs for assessment of nasal congestion and decongestion by closure of the posterior nares with a rubber ball and measuring intranasal pressure from the anterior nares. A reduction in intranasal pressure indicating decongestion has been reported following the intravenous injection of the following drugs: amphetamine, cyclopentamine, epinephrine, ephedrine, mephentermine, methamphetamine, methylaminoheptane, phenylpropanolamine, phenylephrine, *p*-(2-methylaminopropyl)phenol (Pholedrine), and pseudoephedrine.[6-11] There is no information on the ability of eight other currently used sympathomimetic drugs to reduce nasal resistance in man or nasal pressure in dogs.

The experiments that are being reported consist of adapting Jackson's method to the comparative study of effects of drugs on nasal pressure, as well as on vascular resistance of the bed supplied by the common carotid artery in the dog. A rotameter has been inserted into one carotid to measure arterial blood flow. Although most of the drugs studied cause reduction of both measurements (nasal decongestion and carotid vasoconstriction), the relative intensities of both responses are different and some drugs can even cause a paradoxical increase of one or both (nasal congestion and/or carotid vasodilatation).

Submitted for publication Aug. 11, 1958.

Departments of Pharmacology, University of Pennsylvania School of Medicine and Wellcome Research Laboratories.

Methods

Dogs under pentobarbital anesthesia were used (15-25 mg/kg. I. V.). The trachea was routinely cannulated for spontaneous or artificial ventilation with a Starling Ideal pump. One femoral artery was cannulated for recording blood pressure by means of a Statham strain gauge. A recording rotameter[12] was inserted into one common carotid to measure blood flow following intravenous injection (via femoral vein) and intraarterial injection (by direct puncture of the connecting rubber tube between the rotameter and the distal portion of the carotid) of the drug. The nasopharynx was closed with a rubber ball; the nasal cavity was filled with isotonic saline solution, and the nasal orifices were connected to individual Statham transducers for recording intranasal pressures. Sodium heparin (10 mg/kg.) was routinely used as the anticoagulant.

The sympathomimetic drugs were used in aqueous solution such that the total volume injected intra-arterially did not exceed 0.1 ml. The generic names and synonyms (in parentheses) of the drugs used are as follows: cyclopentamine (Clopane), ephedrine sulfate, epinephrine (Adrenalin), 3, 4-dihydroxyphenylethylmethylamine (Epinine), hydroxyamphetamine hydrobromide (Paredrine), levarterenol bitartrate (Levophed) mephentermine sulfate (Wyamine), methamphetamine (Methedrine), methoxamine (Vasoxyl), naphazoline (Privine), phenylephrine (Neo-Synephrine), phenylpropanolamine (Propadrine), phenylpropylmethylamine (Vonedrine), pseudoephedrine (Sudafed), *p*-hydroxyphenylmethylaminoethanol (Synephrin, Sympatol) and tuaminoheptane sulfate (Tuamine).

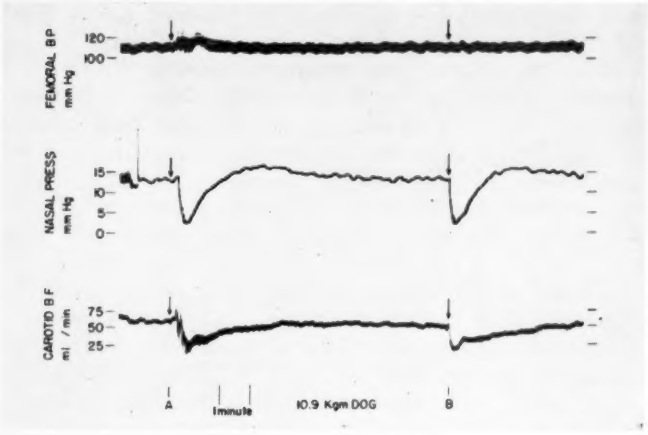


Fig. 1.—Epinephrine (*A*) intravenously 1μg. per kilogram and (*B*) intracarotid 0.1μg. Note the immediate fall in nasal pressure (shrinkage) followed by a rise (after congestion). The reduction in carotid blood flow following intracarotid injection represents vasoconstriction because arterial pressure remains constant.

All the 16 compounds were used as hydrochloride salts, except where indicated otherwise.

Results

The sympathomimetic drugs that are used clinically as nasal decongestants were investigated. The list includes epinephrine, which was used as the standard drug for comparison, and 15 other drugs listed below under three groups based on their comparative effects on nasal pressure and carotid blood flow.

Epinephrine.—The usual response following the intravenous injection of 1μg/kg. of epinephrine consists of a rise in femoral blood pressure and a biphasic response in the carotid blood flow, initial rise followed by a fall. The initial rise in flow is due to the rise in arterial pressure at a time when the drug has not reached the branches of the carotid. This happens in a few seconds, and flow is then reduced, in spite of the continued rise in arterial pressure. The reduction in flow is a manifestation of the local constriction of the carotid bed. Nasal pressure is simultaneously reduced with flow (decongestion), but after about two minutes there is complete recovery to the control level and even a slight rise (congestion). This after-congestion occurs at a time when carotid flow is reduced so that both are altered independently (Fig. 1*A*).

The intracarotid injection of epinephrine (0.1μg. to 1.0μg. total) causes a similar

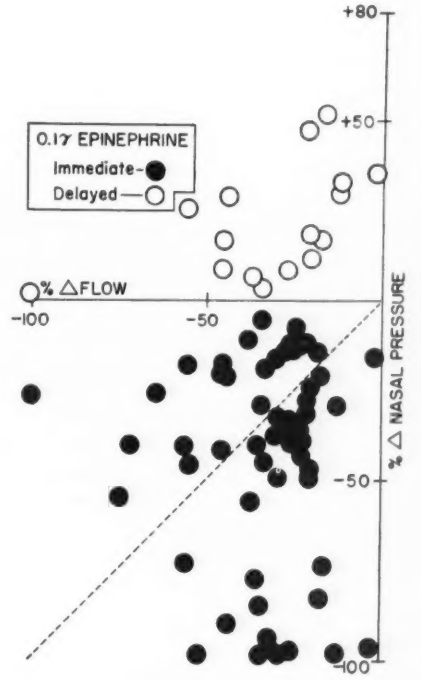


Fig. 2.—Summary of intracarotid injection of epinephrine. Each point represents per cent change in flow (on the abscissa) and per cent change in nasal pressure (on the ordinate). The empty points above the horizontal axis indicate delayed congestion. The dark points above the diagonal line represent results in which the reduction in carotid flow is more intense than reduction in nasal pressure. The reverse is true for the points below the diagonal line.

response because the dose is not large enough to affect the systemic arterial pressure. Carotid blood flow and nasal pressure are instantaneously reduced, but after about two minutes, nasal after-congestion appears at a time when flow is still reduced (Fig. 1*B*). It is safe to interpret the reduction in blood flow to indicate vasoconstriction of the carotid vessels because blood pressure remains essentially unchanged.

A summary of the results obtained from 27 dogs is represented in Figure 2. Each dog was subjected to two intracarotid injections, one at the beginning and the other at the conclusion of the experiment, and the characteristics of the response did not alter in spite of the injections of other sympathomimetic drugs between both. The range of control blood flows was from 30 to 125 ml. per minute, and the immediate effect following epinephrine injection ranged from −20% to −100%. The control nasal pressure ranged from 5 to 25 mm. Hg, with a reduction ranging from 5% to 100%. The absolute level of nasal pressure varied among different dogs and was a function of the amount of saline introduced into the nose. The fact that 22 of the 54 injections showed a less intense per cent reduction of nasal pressure than of flow and that 32 showed a more intense reduction is probably related to the preexisting level of nasal pressure, and does not mean a difference in vascular sensitivity of the carotids and the nose. For each individual dog, the comparative intensity of reduction in flow and resistance remained essentially the same throughout the experiment, i. e., the per cent reduction in nasal pressure was either greater (below diagonal line of Fig. 2) or less (above diagonal) than per cent change in flow.

The phenomenon of after-congestion was not uniformly seen following all intracarotid injections of epinephrine. Only 15 out of 54 injections showed a rise in nasal pressure (by 5%-50%) at a time when flow was still reduced. Its presence or absence was not related to the absolute level of nasal pressure, or to the insertion of the rotameter. The omission of the rotameter in three dogs showed the absence of nasal congestion in one dog at varying levels of nasal pressure.

The most interesting difference between nasal pressure and carotid flow is that nasal pressure can recover to the control level or can even increase at a time when carotid flow is still reduced by epinephrine. There are two possible interpretations for this phenomenon. (*a*) That the difference in methods accounts for the greater sensitivity of nasal pressure recordings than flow measurements. This possibility can be dismissed because acute alterations in blood volume by bleeding or transfusion caused a simultaneous decrease and increase respectively of both measurements. Furthermore, when isoproterenol (a sympathomimetic vasodilator) is used, there is a simultaneous increase of both. (*b*) That the nasal vessels behave differently from the other branches of the common carotid. This appears to be the more likely interpretation because the electrical stimulation of the central end of the cut cervical vagosympathetic trunk with threshold strength would cause a reduction in nasal pressure with minimal effect on blood flow, but the use of stronger currents would cause an additional reduction in carotid blood flow. The sympathetic vasoconstrictors to the nose are more sensitive to electrical stimulation than those supplying the other carotid branches. Finally, the results from some drugs (described below) show a difference in intensity of reduction in nasal pressure and carotid flow, and others show an opposite directional change of both.

Epinine, Ephedrine, Pseudoephedrine, Methoxamine, Phenylpropanolamine, and Synephrin.—These amines are similar to epinephrine in respect to the qualitative changes in femoral blood pressure, nasal pressure, and carotid blood flow following intravenous or intracarotid injections. However, a secondary congestion of the nose was never encountered, indicating that none of these drugs have vasodilator properties and this is supported by studies on other beds reported elsewhere.[11]

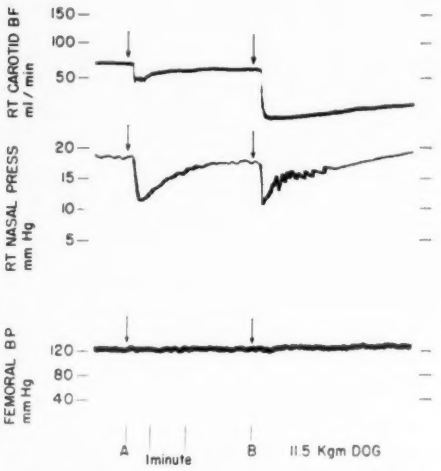


Fig. 3.—Intracarotid injections of (*A*) epinephrine 0.1μg., and (*B*) methoxamine 100μg. Note that although both injections cause an equivalent reduction in nasal pressure; the reduction in flow by methoxamine is more intense and more prolonged than that induced by epinephrine.

The difference in intensity of the reduction in flow and nasal pressure is unique for this group, which is exemplified in Figure 3. The intracarotid injection of 100μg. (total dose) of methoxamine causes a fall in nasal pressure equivalent to that produced by a prior injection of epinephrine, yet the carotid vasoconstriction by methoxamine is more intense. On the basis that the reduction in nasal pressure and flow by a standard injection of 0.1μg. epinephrine is expressed as −100%, the results of the following intracarotid injections have been compared: Epinine (10μg.), ephedrine, pseudoephedrine, methoxamine, phenylpropanolamine (each 100μg.) and Synephrin (1,000μg.). The injection of each drug causes reduction in carotid flow and nasal pressure, either more intense or less intense than that produced by epinephrine. Almost all of the results are characterized by a greater per cent change in flow than the per cent change in nasal pressure, indicating that carotid vasoconstriction is more intense than nasal vasoconstriction.

Although the effects of pseudoephedrine derived from five dogs are in line with

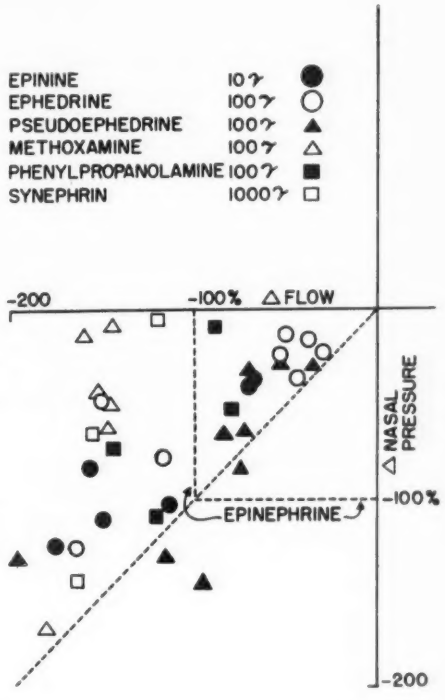


Fig. 4.—Summary of intracarotid injections based on epinephrine response as −100% for flow (abscissa) and −100% for nasal pressure (ordinate). Note that all drugs except pseudoephedrine cause a more intense reduction in flow as compared to the accompanying reduction in nasal pressure.

those just described (above diagonal line of Fig. 4) three dogs behaved differently (below diagonal line). The latter response is represented in Figure 5, which shows that the reduction in flow by pseudoephedrine is equal to that by epinephrine, yet reduction in nasal pressure by pseudoephedrine is greater. The reason for the difference in results obtained from the eight dogs was not explored because repetition of pseudoephedrine injections led to tachyphylaxia, which has been encountered in other organs.[13]

Levarterenol, Phenylephrine, Naphazoline, Cyclopentamine, Hydroxyamphetamine, Tuaminoheptane.—The relatively more intense nasal vasoconstriction seen in some dogs following the injection of pseudoephedrine was more consistently observed when

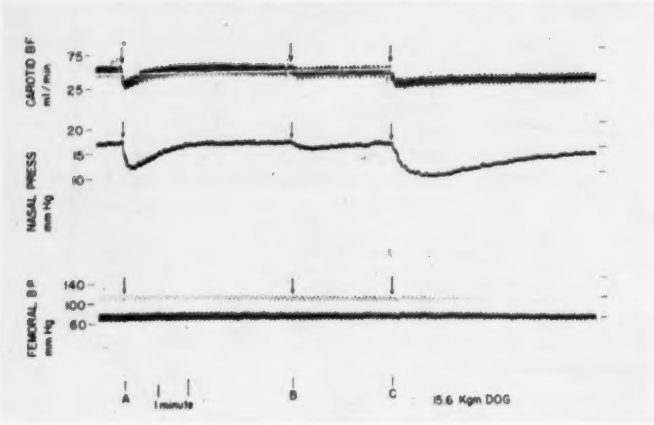


Fig. 5.—Intracarotid injections of (*A*) epinephrine 0.1μg.; (*B*) pseudoephedrine 10μg.; (*C*) pseudoephedrine 100μg. The larger dose of pseudoephedrine caused an equivalent reduction in carotid blood flow compared with epinephrine, but the reduction in nasal pressure is more intense and more prolonged.

six other drugs were injected into the carotid artery. A typical response is represented in Figure 6, in which injections of tuaminoheptane and epinephrine cause equivalent reduction in nasal pressure, but the effect on carotid flow by tuaminoheptane is less than that by epinephrine. The drugs were similarly injected in the following total doses: levarterenol (0.1μg.-1μg.), phenylephrine (1μg.-10μg.), naphazoline (10μg.), cyclopentamine (100μg.), hydroxyamphetamine and tuaminoheptane (both 1,000μg.). The results are summarized in Figure 7. Compared with the standard response to epinephrine, the injection of this group of drugs causes a reduction in nasal pressure that is more intense than the corresponding reduction in carotid flow. The phenomenon of after-congestion was not observed following either intracarotid or intravenous injection.

Methamphetamine, Mephentermine and Phenylpropylmethylamine. — The results have been variable when each one is injected in 1 mg. total dose (Fig. 8). A combined reduction of nasal pressure and carotid flow was encountered, but two other types of response were observed. One type is a simultaneous increase in flow and pressure indicating a combined dilatation, but this is often temporary. After about a minute, either one or both measurements may recover completely or even become reversed. The other type is simpler to analyze and

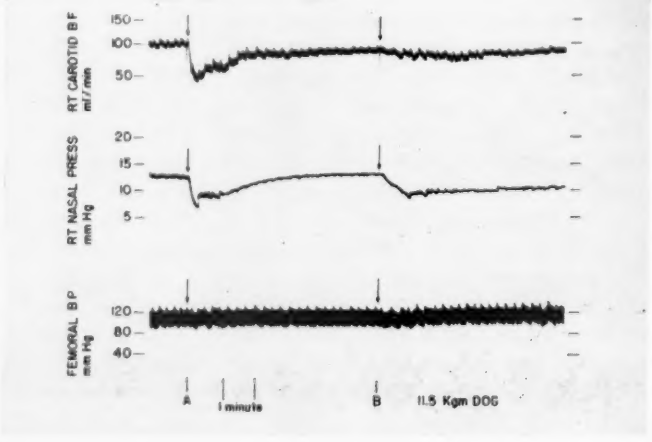


Fig. 6.—Intracarotid injections of (*A*) epinephrine 0.1μg.; (*B*) tuaminoheptane 1,000μg. The injection of the latter causes a significant reduction in nasal pressure but no effect on flow.

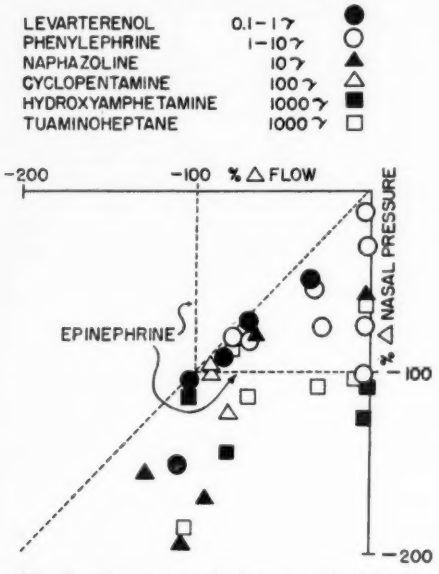


Fig. 7.—Summary of intracarotid injections based on epinephrine response as —100% for flow (abscissa) and —100% for nasal pressure (ordinate). All drugs cause a more intense reduction in nasal pressure than in flow.

consists of a simultaneous increase in flow but reduction in nasal pressure (Fig. 9). This particular combination is the one referred to in the above paragraph as indicating that nasal pressure may change in direction opposite to that of carotid blood flow. The simplest explanation for the varied results is as follows: The components of the common carotid artery respond in different ways to methamphetamine, mephentermine, and phenylpropylmethylamine. At least one component can be constricted but the others can be dilated. Their individual sensitivities are not predictable, so total flow would either increase or decrease or both. The ultimate identification of the vessels that are either dilated or constricted is not possible with the technique utilized presently. Until this is done, it is not possible to specifically state if the blood vessels of the nasal mucosa are actively constricted by these drugs or passively constricted because of diversion of blood flow to the dilated portions. Attempts to isolate the nasal arterial branches for injection of drugs failed because of the necessity to use anticoagulants for the rotameter.

Comment

The most significant observation of the above experiments is the difference in sensitivity of nasal vessels from other branches of the common carotid. The nasal decongesting drugs may be divided into three groups. One group (exemplified by levarterenol, phenylephrine, naphazoline, cyclopentamine, hydroxyamphetamine, and tuaminoheptane) appears to have a more selective nasal decongestion. A second group (Epinine, ephedrine, methoxamine, phenylpropanolamine, and Synephrin) appears to have a less selective nasal decongestion. Overlapping both of these groups are a number of drugs (pseudoephedrine, methamphetamine, mephentermine, and

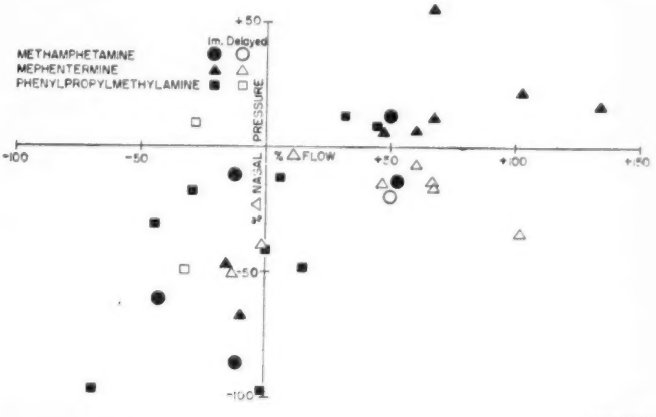


Fig. 8.—Summary of intracarotid injections, not corrected for epinephrine response as in Figures 4 and 7.

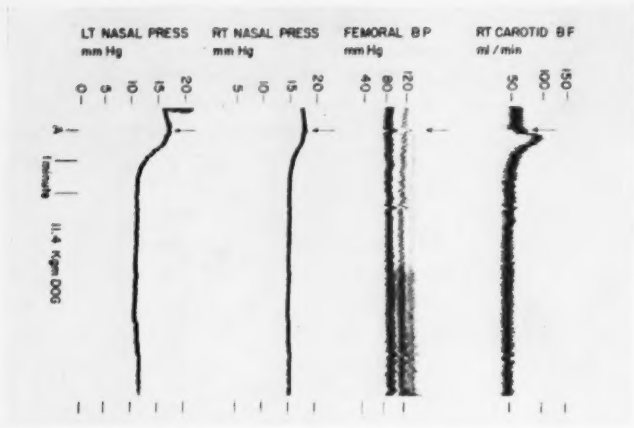


Fig. 9.—Intracarotid injection of methamphetamine 1 mg. The immediate increase in flow is accompanied by a reduction in nasal pressure.

phenylpropylmethylamine), including epinephrine, upon which the first two groups are based.

It would be very desirable to obtain confirmation of local nasal vasoconstriction by a method other than the use of carotid injection. The most logical way would be to simulate the clinical practice of applying the drugs locally on the nasal membrane, and measure blood flow to the nasal mucosa appropriately. It is possible that local application would stimulate some receptors on the mucosal surface that would contribute to decongestion by reflex vasoconstriction, and this could be missed by giving the drugs into the blood supply of the nose. This approach was not pursued in dogs because information derived directly from human subjects would be more pertinent to the problem.

The results presently described may have some bearing on the mechanism of nasal decongestion induced by the oral administration of pseudoephedrine, ephedrine, methamphetamine, phenylpropanolamine, hydroxyamphetamine, and phenylephrine.[14] If one assumes that an effective concentration in the blood is reached, nasal decongestion would be expected to occur similar to the reduction in intranasal pressure encountered in dogs following injection of drugs. Although the selectivity within the carotid bed reported above would favor some drugs (levarterenol, phenylephrine, naphazoline, cyclopentamine, hydroxyamphetamine, and tuaminoheptane), there are other factors to consider, such as oral absorption, cardiac stimulation, pressor action, and depression of the central nervous system. Each one of these drugs unfortunately has powerful cardiovascular actions. Stupor arising from excessive amounts of naphazoline has been reported.[15,16] It would be highly desirable if the comparative study reported above were extended to human subjects in which drugs are given orally to relieve nasal congestion.

Summary and Conclusions

Measurements of intranasal pressure and carotid blood flow in anesthetized dogs revealed some interesting differences among the following sympathomimetic drugs injected directly into the common carotid. Epinephrine caused carotid constriction and nasal decongestion followed by after-congestion. One group of drugs (3,4-dihydroxyphenylethylmethylamine[Epinine], hydroxyphenylmethylaminoethanol [Synephrin], ephedrine, methoxamine, and phenylpropanolamine) caused carotid vasoconstriction as intense as epinephrine, but a slighter degree of reduction in nasal pressure. Another group (levarterenol, phenylephrine, naphazoline, cyclopentamine, hydroxyamphetamine, tuaminoheptane) caused a relatively more intense reduction in nasal pressure as compared with carotid

vasoconstriction. Pseudoephedrine manifested activity overlapping both groups; on the average it caused almost equal reduction of flow and nasal pressure. Methamphetamine, mephentermine, and phenylpropylmethylamine caused a decrease as well as an increase in nasal pressure, indicating that the nasal pressure changes may arise from varied effects on the carotid branches which have not yet been identified.

Laboratory of Pharmacology, University of Pennsylvania School of Medicine and Wellcome Research Laboratories.

REFERENCES

1. Uddstromer, M.: Nasal Respiration: A Critical Survey of Some Current Physiological and Clinical Aspects on the Respiratory Mechanism with a Description of a New Method of Diagnosis, Acta oto-laryng. Supp. 42, pp. 3-146, 1940.

2. Seebohm, P. M., and Hamilton, W. K.: A Method for Measuring Nasal Resistance Without Intranasal Instrumentation, J. Allergy 29:56-59 (Jan.) 1958.

3. Butler, D. B., and Ivy, A. C.: Effects of Nasal Inhalers on Erectile Tissues of the Nose; Quantitative Studies, Arch. Otolaryng. 38:309-317 (Oct.) 1943.

4. Jackson, D. E.: Experimental Pharmacology and Materia Medica, Ed. 2, St. Louis, C. V. Mosby Company, 1939.

5. Jackson, D. E.: Experimental and Clinical Observations Regarding the Physiology and Pharmacology of the Nose, Ann. Otol. Rhin. & Laryng. 51:973-994 (Dec.) 1942.

6. Jackson, D. E.: The Pharmacologic Action of 2-Methyl-Aminoheptane (EA-1), J. Lab. & Clin. Med. 29:150-167 (Feb.) 1944.

7. Shirkey, H. C., and Jackson, D. E.: Experimental and Clinical Observations Regarding the Nasal Vasoconstrictor Action of Oenethyl and EA-83, Ann. Otol. Rhin. & Laryng. 55:859-871 (Dec.) 1946.

8. Chen, K. K.: Aliphatic Versus Aromatic Amines as Vasoconstrictors, Ann. Otol. Rhin. & Laryng. 57:287-298, (June) 1948.

9. Roth, L. W.; Shemano, I.; Morphis, B. B., and Richards, R. K.: Studies on the Comparative Analeptic and Circulatory Activities of d- and l-Desoxyephedrine, J. Pharmacol. & Exper. Therap. 106:412, (Dec.) 1952.

10. Eckfield, D. K.; Abell, L. L., and Seifter, J.: Pharmacologic Properties of Mephentermine—a Sympathomimetic Amine, J. Am. Pharm. A. (Sc. Ed.) 43:705 (Dec.) 1954.

11. Aviado, D. M., Jr.; Wnuck, A. L. and de Beer, E. J.: Cardiovascular Effects of Sympathomimetic Bronchodilators: Epinephrine, Ephedrine, Pseudoephedrine, Isoproterenol, Methoxyphenamine and Isoprophenamine, J. Pharmacol. & Exper. Therap. 122:406-417 (March) 1958.

12. Shipley, R. E., and Wilson, C.: An Improved Recording Rotameter, Proc. Soc. Exper. Biol. & Med. 78:724-728 (Oct.) 1951.

13. Chen, K. K., and Schmidt, C. F.: Ephedrine and Related Substances, Medicine 9:1-117 (Feb.) 1930.

14. Physicians Desk Reference, to Pharmaceutical Specialties and Biologicals, Oradell, N. J. Medical Economics, Inc., pp. 748, 757, 772, 780 and 784; also p. 9 of May. Supp., 1958.

15. Arvola, A.: Rhinitis Medicine as a Cause of Poisoning, Ann. med. int. Fenniae 36:228-231, 1947.

16. House, L. R., and Carey, W. C.: Constitutional Effects from the Use of Sympathomimetic Drugs as Nasal Medication in Children: Report of Case of Privine Toxicity, Laryngoscope 58: 1294-1298, 1948.

Audiology Section

JOSEPH SATALOFF, ASSOCIATE EDITOR

The Efficiency of Limited Frequency Audiometric Screening in a School Hearing Conservation Program

CLIFTON F. LAWRENCE, M.A., and WALLACE RUBIN, M.D., New Orleans

Introduction

During recent months, studies have been reported relative to a new method for audiometric screening which has been designated as "limited frequency" testing.[4-7] It has been proposed that this method be substituted for present sweep-check and group test methods used in school hearing conservation programs.

As recommended by House and Glorig, the test would consist simply of the presentation of a 2,000 cycle and a 4,000 cycle tone at a specified intensity (presumably 15 or 20 db.) to the ear under test. It has been further stated that approximately the same results can be obtained by presenting only the 4,000 cycle tone. At least one manufacturer has brought out a small transistorized instrument for use in "limited frequency" testing.

If the efficiency of this type of test could be established, there would be many obvious advantages in its use in preference to the routine sweep-check test, and possibly in preference to present group tests. Particularly in school hearing conservation programs, where training of personnel and cost and maintenance of equipment are important factors, the development of a simple, rapid, and economical test would solve many practical problems. However, before any such method could be adopted it would be necessary to obtain some definitive evidence that the test would actually result in selection of cases with at least the degree of efficiency found in presently used standard screening methods. The present study was planned to obtain preliminary data which would indicate whether or not this "limited frequency" method would meet such a criterion.

Submitted for publication Sept. 26, 1958.

Presented at the American Speech and Hearing Association meeting, New York, Nov. 17, 1958.

Administrative Audiologist, Louisiana State Department of Health (Mr. Lawrence). Otologist (Dr. Rubin).

Procedures

Three groups of children were involved in the study. Group I consisted of 1,000 school children in Grades 1 through 12 enrolled in the parochial schools in New Orleans. Group II consisted of a different group of 1,000 children with the same grade distribution and school enrollment. Group III consisted of 536 children for whom case records were available, and who had been examined at otological clinics under the State of Louisiana Hearing Conservation Program. Children in Groups I and II were all tested by the same audiometric technician, with testing done in a mobile sound-treated unit which was moved from school to school during a six-week period.

Children in Group I were each given a "limited frequency" test at 15 db. utilizing frequencies 2,000 and 4,000. The instrument used was an Ambco "Oto-Chek" Model 700. Following this test each child was given a routine sweep-check test at 15 db. using frequencies 250, 500, 1,000, 2,000, 4,000, and 8,000. The instrument used for this testing was a Beltone Model 10-A pure-tone audiometer. Both instruments were checked for correct calibration prior to and following the testing, using an Allison Model 300 Audiometer Calibration Unit. Appropriate measures were used to insure consistent procedure throughout the testing, and to minimize technician and equipment variables.

Children in Group II were given only a sweep-check test at 15 db. at frequencies 250, 500, 1,000, 2,000, 4,000, and 8,000.

Children in Group III had originally been selected by a routine sweep-check test (250 through 8,000 cycles) in various schools throughout the state. Data available included a complete pure-tone audiogram and otological examination for each child.

As noted, the 2,000 children screened in the New Orleans schools were divided into two numerically equal groups. For the first group, the "Oto-Chek" was used to check frequencies 2,000 and 4,000 prior to the sweep-check; in the second group the "Oto-Chek" was not used, but notation was made as to each frequency the child failed to respond to at 15 db. This procedure was used to gain information as to the test-retest situation, and to determine whether any significant difference might result when tones were presented in relative isolation (with the "limited frequency" method) and then as part of a sequence of tones (with the routine sweep-check).

Method of Analysis

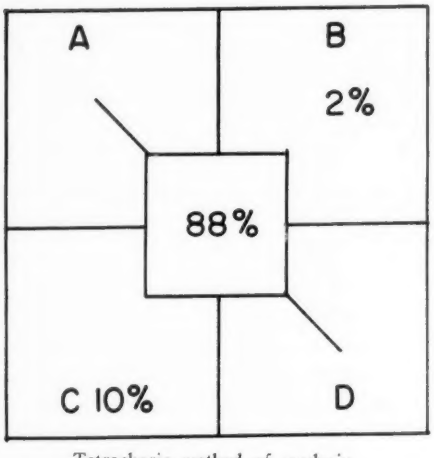
Different investigators[2,3,9] have proposed use of a "tetrachoric table" for analysis of data when comparing group tests with individual tests. Data are recorded in four categories, or cells, as follows:

Cell "A"—Percentage of children "passing" both group and individual tests

Cell "B"—Percentage of children "passing" group and "failing" individual test

Cell "C"—Percentage of children "failing" group and "passing" individual test

Cell "D"—Percentage of children "failing" both tests.



Tetrachoric method of analysis.

DiCarlo and Gardner[3] have suggested the criteria illustrated in the Figure.

An effective group test should have a hypothetical distribution of agreements and disagreements as indicated in the diagram. Children either passing or failing both tests should make up a minimum of 88% of the total (Cell "A" plus Cell "D"), while a maximum of 2% should be "missed" by the group test (Cell "B") and a maximum of 10% should be unnecessarily referred by the group test (Cell "C"). Obviously, the nearer cells "B" and "C" are to zero and the nearer the combined percentage in cells "A" and "D" approaches 100% the closer the agreement between the tests being compared.

Since group tests and the "limited frequency" test under investigation are identical as to purpose, the tetrachoric method of analysis was selected for data in this study. By the use of this method both limited frequency and group test data can be directly compared with individual sweep-check results. However, even with a test meeting the above hypothetical criteria, we might find that considerable disagreement exists between the two tests as to specific cases selected. We need, in other words, an additional measure to show us what proportion of the group failing the sweep-check test also fail the group (or limited frequency) test. A percentage figure to indicate this agreement can be obtained simply by dividing the percentage in Cell "B" (those cases missed by the test under investigation) by the sum of the percentages in Cells "B" and "D" (those cases selected by the test used as a criterion) and subtracting the obtained quotient from 100. This "percentage of agreement" has therefore been included in analysis of data in the present study.

Analysis

Test-retest reliability. In Group I a total of 15 children (1.5%) either failed the 2,000 or 4,000 cycle tone on the "Oto-Chek" and subsequently heard it on the sweep-check test, or heard the tone on the

TABLE 1.—*Criterion: Routine Sweep-Check at 250-8,000 Cycles, with Failure Defined as Nonresponse to Two or More Frequencies in One Ear at 15 Db.*

Test	Passed Both Trial Test & Criterion	Failed Both Trial Test & Criterion	Passed or Failed Both	Passed Trial Test; Failed Criterion	Failed Trial Test; Passed Criterion	Disagreement	Agreement
	Cell "A"	Cell "D"	Cell "A" & Cell "D"	Cell "B"	Cell "C"	"B" / "B" & "D"	
Group I							
4,000 cycles	93.5%	3.9%	97.4%	1.6%	1.0%	30%	70%
4,000/2,000	93.5%	4.1%	97.6%	1.3%	1.1%	24%	76%
Group II							
4,000 cycles	91.1%	4.9%	96.0%	2.5%	1.5%	34%	66%
4,000/1,000	91.4%	5.2%	96.6%	1.9%	1.5%	27%	73%
4,000/500	91.1%	6.6%	97.7%	0.7%	1.6%	10%	90%
DiCarlo and Gardner "best" group test	87.5%	6.3%	93.8%	0.8%	5.3%	11%	89%
"Modified" sweep check at 500-4,000 cycles	91.6%	7.0%	98.6%	0.4%	1.0%	5%	95%

"Oto-Chek" and subsequently failed it on the sweep-check test. Various investigators [1] have demonstrated that approximately 10% of any given group of children will show a ±5 db. variation in repeat pure-tone threshold measurements. Therefore, it was felt that the 1.5% incidence obtained in this group could be interpreted as indicating that acceptable test-retest reliability exists. Since "practice effect" did not seem to appreciably effect results, it was decided that analysis of the relationships between individual frequencies and total test performance would be justified.

Table 1 summarizes the results obtained when various methods are compared with the routine sweep-check methods. These methods are the following: the "Oto-Chek" test at 2,000 and 4,000 cycles (Group I); specific combinations of tones (Group II); the "best" group test recommended by DiCarlo and Gardner, and a modified sweep-check using 500 through 4,000 cycles. The routine sweep-check in this study is specifically defined as presentation of tones 250, 500, 1,000, 2,000, 4,000, and 8,000 to each ear at 15 db. with "failure" defined as nonresponse to two or more of these tones in one ear, in accordance with recommendations of the American Academy of Ophthalmology and Otolaryngology.[12]

Inspection of the data will disclose several interesting points:

1. Except for results with 4,000 cycles alone in Group II, all "limited frequency" combinations would meet hypothetical criteria proposed by DiCarlo and Gardner for an effective screening test.

2. The number of unnecessary referrals is small (1.5% at most) when compared with the "best" group test combination (5.3%).

3. Screening at only 4,000 cycles shows only 66% to 70% agreement with routine sweep-check testing.

4. Screening at 4,000 and 2,000 cycles, combined, gives some increase in percentage of agreement (76%).

5. Screening at 4,000 and 500 cycles, combined, shows the best (90%) agreement with routine sweep-check testing, and compares favorably with the "best" group test combination.

6. There is close agreement (95%) when frequencies 500, 1,000, 2,000, and 4,000 are considered as a form of modified sweep-check in Group II and this combination of tones is compared with the routine sweep-check. This close relationship, along with other considerations, led to the further comparison of specific frequency combinations with this modified sweep-check test.

Table 2 gives results when specific frequency combinations are compared with this experimental criterion. The experimental criterion is a modified sweep-check at 15 db. using frequencies 500, 1,000, 2,000, and 4,000. Failure is defined as nonresponse to any one of these tones in either ear. This

TABLE 2.—*Criterion: Modified Sweep-Check at 500-4,000 Cycles, with Failure Defined as Nonresponse to Any Single Frequency at 15 Db.*

Test	Passed Both Trial Test & Criterion	Failed Both Trial Test & Criterion	Passed or Failed Both	Passed Trial Test; Failed Criterion	Failed Trial Test; Passed Criterion	Disagreement	Agreement
	Cell "A"	Cell "D"	Cell "A" & Cell "D"	Cell "B"	Cell "C" *	"B" / "B" & "D"	
Group I							
4,000, cycles	94.1%	4.4%	98.5%	1.1%	0.4%	20%	80%
4,000/2,000	93.9%	4.9%	98.8%	0.8%	0.4%	14%	86%
Group II							
4,000/1,000	91.5%	6.8%	98.3%	0.8%	0.0%	20%	80%
4,000/500	91.5%	8.3%	99.8%	0.2%	0.0 %	2%	98%

* Cell "C," in this comparison, represents test-retest error. This is indicated as zero in Part II for 1,000 & 500 cycles, but could actually be expected to correspond approximately with the 0.4% in Part I. It would not affect cells B or D.

comparison also yields some interesting information:

1. Screening at 4,000 cycles only agrees more closely with modified sweep-check testing than it does with routine full-range sweep-check testing. (80% compared with 66%-70%).

2. Screening at 4,000 and 2,000 cycles combined or at 4,000 and 1,000 cycles combined would not materially increase agreement with modified sweep-

3. Screening at 4,000 and 500 cycles combined shows extremely close agreement with modified sweep-check testing (98% agreement).
check test over screening at 4,000 cycles alone.

Table 3 gives results for 536 cases referred to otological clinics relative to the percentage of medically significant cases which would have been referred to such clinics if a "limited frequency" screening test had been used initially. These data must be interpreted very conservatively, of course, in view of the many uncontrolled factors in the audiometric testing and clinical examination. Of the 536 cases seen at clinic, a total of 68 (12.7%) were found to have either normal hearing with no ear pathology or an insignificant loss for which no medical recommendations were made. These cases were classified as "medically nonsignificant," and the balance (87.3%) were classified as having "medically significant" losses. "Medically significant" cases, for purposes of this study, are those cases which could benefit from medical or surgical therapy for restoration of hearing, or those cases where rehabilitative care is necessary for more efficient use of residual hearing. Assuming that a high test-retest reliability exists, the table indicates the percentage of cases in both classifications which would have been referred through initial screening with various "limited frequency" combinations. In effect, results from this type of comparison would seem to indicate that screening at 250 through 8,000 cycles (which was the method used in selecting all 536 cases initially) may *not* be the most efficient method for finding cases in a school hearing conservation program. The percentage of "missed" medically significant cases was only 0.6% with the 4,000 and 500 cycle combination. However, this combination was the least effective in limiting the referral of medically nonsignificant cases.

TABLE 3.—*Percentage of Medically Significant Cases*

Frequency (s)	% Medically Significant Cases Which Would Have Been Referred	% Medically Nonsignificant Cases Which Would Have Been Referred
4,000 at 15 db.	97	52
4,000 & 500 at 15 db.	99.4	65
4,000 & 1,000 at 15 db.	98.7	59
4,000 & 2,000 at 15 db.	98.3	56

Screening at 4,000 cycles only "missed" 3% of the medically significant cases but merits consideration as the most effective means of limiting the selection of medically nonsignificant cases.

Comment

It should be pointed out that almost all investigators who have published data relative to the efficiency of screening tests of one type or another have made the basic assumption that the pure-tone audiometric test is the criterion with which all such screening tests should be compared. Recently, some studies have been reported which are critical of this concept. Withnell,[10] for example, in a study of case records in England, found that approximately 10% of cases "passing" a routine audiometric sweep-check were found to have a "clearly positive" ENT history and/or middle ear pathology. Our data for Group III, on the other hand, indicate that 12.7% of cases selected on the basis of audiometric findings were of no medical significance. Yankauer et al.,[11] in addition to pointing out the lack of uniformity in standards for audiometric testing and criteria for selection of failures, state that "although the diagnosis and significance of severe or moderate loss of hearing seems well standardized, the same cannot be said about minor degrees of hearing loss. The problem calls for longitudinal studies over a long period of time, correlating audiological, medical, and educational observations, before definitive statements can be made."

We agree that more intensive investigation is in order. Certainly a school hearing conservation program which is oriented toward preventive medicine needs to be directed toward finding, with the greatest possible efficiency, those children who will benefit from the services that can be provided. At the present time, there would seem to be ample reason for questioning the assumption that the routine sweep-check test is the ultimate method for case selection in such programs. There is a specific need for otoaudiologic investigation which could determine the existence of ear pathology in school-age children as a primary consideration, rather than limiting otological studies to those children already screened out in one way or another by audiometric testing.

However, until definitive evidence is available as to the testing method of choice for selection of medically significant cases in hearing conservation programs, the routine sweep-check method will undoubtedly continue to be used as the criterion.

A considerable amount of investigation remains to be done, if we are to reach general agreement regarding the testing method of choice for selection of medically significant hearing losses in hearing conservation programs. Evidence from our present studies indicates that results with routine full-range sweep-check testing correlates favorably with results obtained with certain types of "limited frequency" or "modified sweep-check" testing. A "limited frequency" test using 4,000 and 500 cycles would best meet reasonable criteria for agreement with the standard sweep-check test. We recommend that otologists and audiologists responsible for the determination of policy in hearing conservation programs plan continued experimental use of "limited frequency" testing in order that further data can be obtained and reported for study.

305 Baronne St. (12).

REFERENCES

1. Carrell, J., and Gormley, G. J.: A Critical Review of the Literature on the Validity and Reliability of the Audiogram, Speech Monogr. 13: 66, 1946.

2. DiCarlo, L. M., and Gardner, E. F.: The Efficiency of the Massachusetts Pure Tone Screening Test as Adapted for a University Testing Program, J. Speech & Hearing Disorders, 18:175 (June) 1953.

3. DiCarlo, L. M., and Gardner, E. F.: The Efficiency of 3 Group Pure Tone Screening Tests for Public School Children, Exceptional Children, Vol. 24, pp. 351-359, April, 1958.

4. Glorig, A., and House, H. P.: A New Concept in Auditory Screening, A. M. A. Arch. Otolaryng. 66:228 (Aug.) 1957.

5. Glorig, A., and House, H. P.: New, Practical Concept of Hearing Testing for Use of the General Physician, J. A. M. A. 166:1719 (April 5) 1958.

6. House, H. P., and Glorig, A.: A New Concept of Auditory Screening, Laryngoscope, 67:661 (July) 1957.

7. House, H. P.: A New Instrument and Concept for the Rapid Detection of a Possible Hearing Impairment, Tr. Am. Acad. Ophth. 61: 228 (March-April) 1957.

8. Meyerson, L.: Hearing for Speech in Children: A Verbal Audiometric Test, Acta oto-laryng. Supp. 128, 1955-1956.

9. Newby, H. A.: Evaluating the Efficiency of Group Screening Tests of Hearing, J. Speech & Hearing Disorders, 13:236 (Sept.) 1948.

10. Withnell, A.: The Detection of Ear Defects in School Children, Medical M. Officer, 99:5-6 (Jan. 3) 1958.

11. Yankauer, A.; Geyer, M. L., and Chase, H. C.: Comparative Evaluation of 3 Screening Methods for Detection of Hearing Loss in School Children, Am. J. Pub. Health 44:77 (Jan.) 1954.

12. Manual for a School Hearing Conservation Program, Committee on Conservation of Hearing, Tr. Am. Acad. Ophth., Supp., 1951.

Use of the Plastic Needle in Transtracheal Anesthesia

A New Technique

HESTON L. WILSON, M.D., San Diego, Calif.

Use of the plastic needle in transtracheal anesthesia overcomes problems which have beset the transtracheal technique in the past. It was Canuyt[1] who first suggested the method of injecting medication directly into the trachea. He used a short needle inserted through the skin and cricothyroid membrane into the lumen. This method remained popular in Europe for many years before it came to attention in this country. Harken and Salzberg[2] with a series of 1,000 patients, first reported its use here. The technique has been used not only for instillation of topical anesthetic, but for contrast media for bronchography as well.

Several shortcomings of the older technique have been apparent. The first of these is the necessary rapid instillation of anesthetic solution. This is brought about by the severe coughing produced by initial injection of solution. Thus, with a rigid needle through the skin and into the trachea, the solution must be injected quickly to avoid breakage of the needle or unnecessary trauma to soft tissue.

Adriani and Parmley[3] report two cases in a series of 350 instillations in which complications occurred. In one cellulitis of the neck occurred, and in the other the needle was broken. They used a 25-gauge needle. I have had no complications in a series of 250 instillations using the transtracheal technique employing a 21-gauge ordinary steel needle. In spite of this the following limitations made an improved technique desirable. The technique, as it will be described, using the plastic needle, makes needle breakage impossible, and with adequate sterile precautions cellulitis should be negligible.

Moore and Tolan[4] have ably demonstrated the importance of slow application of a toxic anesthetic agent. They show that rapid administration produces a greater number of untoward reactions without improved anesthesia.

Another limitation of the older method is failure to produce thorough anesthesia throughout the tracheobronchial tree. Usually only the larynx and upper trachea were adequately anesthetized, and additional anesthetic solution was required during the course of bronchoscopy. By the oral techniques, little more than adequate anesthesia of the larynx could be obtained. The present technique readily overcomes these limitations.

The plastic needle used is a Rochester plastic needle, manufactured by Rochester Products Company, Rochester, Minn. Either size 16 or 17 gauge is used. This is the needle normally used for vena punctures. It is pictured in Figure 1. A skin wheal is raised over the cricothy-

Fig. 1.—Complete Rochester plastic needle with stylet in place.

Submitted for publication June 28, 1958.

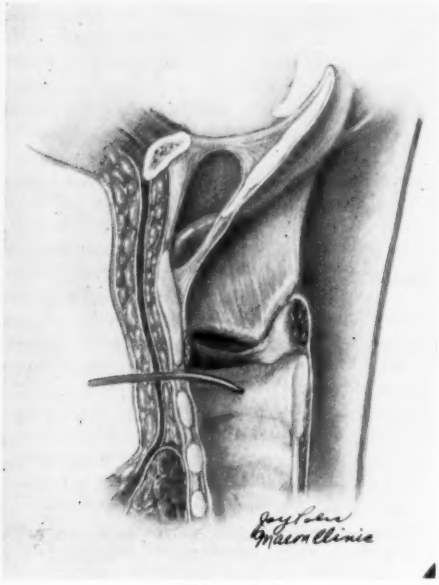


Figure 2
Fig. 2.—Plastic needle in place.

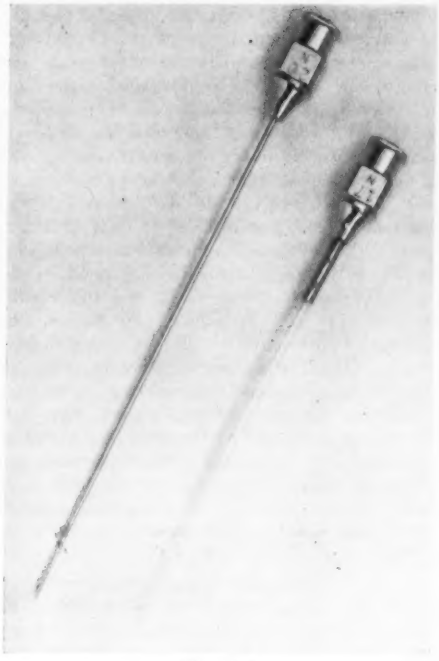


Fig. 3.—Rochester plastic needle with stylet removed.

Figure 3

roid membrane, using procaine 2%. The trocar is then passed through the skin and finally through the cricothyroid membrane as shown in Figure 2. The needle is attached to a syringe containing cocaine 4% at the time the trocar is injected. Air is drawn into the barrel of the syringe to assure presence of the tip within the trachea. The stylet is thereupon removed from the plastic portion of the needle. As the plastic is quite flexible, the traction of the muscular and fascial planes of the neck serves to hold it well in place, and at no time has slippage of the tubing occurred with normal precautions. The patient is then placed in a sitting position. He is asked to lean far to the right, and a few minims of anesthetic solution is instilled. Patient is then inclined to the left, and more of the cocaine is injected into the tracheobronchial tree. A fair amount of coughing is normally produced as each instillation of anesthetic agent is performed. The procedure is done over a period of several minutes, avoiding rapid absorption of the agent. No more than a total of 2 cc. of 4% cocaine is at one time employed. As anesthesia progresses, complete obtundation of the cough reflex is obtained. At this point the plastic tube is removed, and endoscopic manipulation performed. In my hands this procedure has proved highly satisfactory, with anesthesia adequate throughout the tracheobronchial tree.

In bronchography the anesthetic technique is the same, though when thorough anesthesia is secured, the contrast medium is injected through the plastic needle just as was the anesthetic agent. This technique has been found ideal in bronchography, especially as it has been of extreme usefulness in very apprehensive patients in whom bronchography had previously been unsuccessfully attempted without anesthesia.

I have performed bronchoscopy with the polyethylene tube still present within the tracheal lumen. No abnormal tears or other complicating factors have been evident upon performance of this procedure.

Two patients are presented to demonstrate the efficacy of this technique. The first is a 38-year-old white man first hospitalized on Nov. 2, 1957. The patient on that same day had been involved in an automobile accident with multiple injuries. Immediate laparotomy was performed with closure of a rupture of the liver and ligation of the bleeding points in the retroperitoneal area about the pancreatic capsule. There was much blood in the peritoneal cavity. The left lobe of the liver was shattered, with some fragments in the splenic area. Patient's course was difficult, with high temperature and

high pulse rate. Chest x-ray taken on the day of entry was essentially negative. On Nov. 6, with increasing complaint of chest pain and cough, repeat x-rays showed collapse of the right middle lobe and questionable obstruction of the right lower bronchus. At this time bronchoscopy was performed following anesthesia by the above technique. The bronchoscope was passed with ease, and highly adequate anesthesia had been obtained. The right main-stem bronchus was filled with thick mucus, which was also found obstructing the middle lobe on the right side. These areas were suctioned thoroughly, and except for the mucus obstruction, the tracheobronchial tree was found within normal limits. The patient's course, however, was considered unsatisfactory, though the pulmonary complaints were markedly lessened. On Nov. 15 the patient died. At the time of autopsy he was found to have severe bile peritonitis and pancreatitis with lacerations of the liver. The lung was clear except for terminal pneumonia in the lingula.

The next patient was a highly apprehensive 17-year-old white girl with a history of pneumonia four times in the previous four years, and in the previous year she had had three bouts of hemoptysis. The cough persisted and had been productive of foul yellowish sputum. There had been persistent sensation of distress in the right lower chest. Only a recounting of the various x-ray reports would indicate the difficulties encountered in this highly apprehensive adolescent girl.

X-ray on April 16, 1957, reported: "Because of the patient's extremely irritable tracheobronchial system, bronchograms unsatisfactory. Partial filling was obtained in the basilar segments of the right lower lobe which showed no gross evidence of bronchiectasis."

Repeat bronchogram was attempted on April 17, 1957. Report: "Bronchogram was performed with considerable difficulty and only the right side was satisfactorily filled due to inability of the patient to cooperate further. There is cylindrical bronchiectasis of the posterior basilar segment on the right with incomplete filling of the anterior and lateral basilar segment. The middle lobe and upper lobe are within normal limits, as is the superior segment of the lower lobe. No statement can be made due to insufficient filling on the left side." The next report was April 24, 1957. "Left bronchographic studies done under general anesthesia failed to demonstrate bronchiectasis in any of the segments outlined on that side. Nor is there any evidence of bronchial papillomatosis."

Because of persistence of symptomatology, the internist in charge of the patient felt that further bronchographic studies were indicated, and I was called in consultation on Nov. 25, 1957. At this time, when the plastic needle was used in the transtracheal method, the report was as follows: "Bronchography for transtracheal injection of Dionosil results in good filling and demonstrates normal bronchial elements on the left, and also in the right upper and middle lobes. There are prominent cylindrical bronchiectatic changes in the lateral basal segment of the right lower lobe. Slight ectasia in the posterior basal segment. Anterior and medial basal segments appeared to be within normal limits."

It is believed that these two cases adequately illustrate the efficacy of the transtracheal technique, both in anesthesia to the tracheobronchial tree, and in bronchography. It is my impression that this modification of the transtracheal technique will bring the direct instillation of medication into the tracheal lumen to its deserved popularity.

411 Thorn St.

REFERENCES

1. Canuyt, G.: Anesthesia locale en Otorhinolaryngologie, Sc. méd. Bordeaux 41:266-269, 1920.

2. Harken, D. E., and Salzberg, A. M.: Transtracheal Anesthesia for Bronchoscopy, New England, J. Med. 239:383-385, 1948.

3. Adriani, J., and Parmley, J.: Complications Following Transtracheal Anesthesia, Am. J. Surg. 84:11-12, 1952.

4. Moore, D. C., and Tolan, J. F.: Anesthesia for Surgery of the Nose, Pharynx, Larynx, and Trachea, A. M. A. Arch. Otolaryng. 64:275-288, 1956.

5. Bonica, J. J.: Transtracheal Anesthesia for Endotracheal Intubation, Anesthesiology 10: 736-738, 1949.

A Bilateral Posterior Intranasal Pack

An Evaluation of Various Types of Postnasal Packing

SIDNEY H. GIDOLL, M.D., San Francisco

The multiplicity of therapeutic measures offered for a single disease suggests the inadequacy of any one method alone to effect a cure. Similarly, the literature has been replete with a wide variety of suggestions for the control of severe posterior nasal hemorrhage. Here again, the diversity of the types of posterior packs described indicates the dissatisfaction of each successive writer with the effectiveness of the various types of posterior nasal packs available to him up to the time of his report. As a matter of fact, they are all effective when appropriately applied to the specific condition for which each one is best suited.

When one is faced with an emergency situation in which there is a profuse intranasal hemorrhage from a posterior source, the origin of which cannot be located at the time, it may be assumed that the source, either laterally or medially, is a branch of the sphenopalatine artery.

This view is confirmed by Schaeffer,[1] who states that the main intranasal blood supply is from the sphenopalatine artery. Davis[2] similarly writes that epistaxis occurs in a large percentage of cases from the septal branch of the sphenopalatine artery. According to Saunders,[3] posterior nosebleeds usually come from the posterior end of the inferior meatus. Woodruff[4] agrees that the sphenopalatine artery or one of its branches is the bleeding vessel in the great majority of cases.

Submitted for publication Aug. 7, 1958.

Septal Vascular Distribution vs. Lateral Nasal Wall

Even at the risk of appearing elementary, the blood supply of both the nasal septum and of the lateral nasal wall is briefly reviewed for the purpose of illustrating a point in hemostasis. The blood supply of the internal nose is derived indirectly from two different arterial systems, the external carotid artery and the internal carotid artery.

The internal carotid artery supplies the upper and anterior portions of the nose via a branch, the ophthalmic artery, from which are derived the anterior and posterior ethmoidal arteries. The nasal branches of the latter two arteries enter the internal nose at its roof and supply branches to the anterior and superior parts of both the nasal septum and the lateral nasal wall (Figs. 1 and 2). In other words, the arterial distribution on the anterior and superior portions of the septum practically parallels the arterial distribution on the ipsolateral portion of the lateral nasal wall.

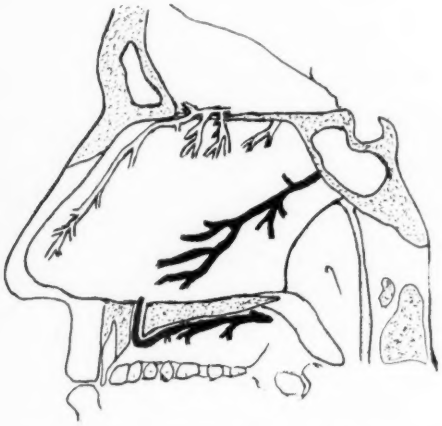


Fig. 1.—Diagrammatic illustration of the submucosal arterial blood supply to the septum; anterior and posterior ethmoidal artery branches are shown in outline form; sphenopalatine artery branches are shown in solid black.

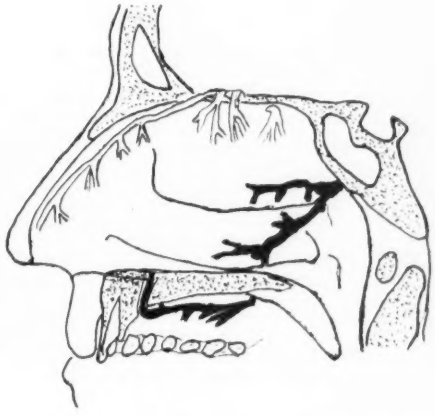


Fig. 2.—Diagrammatic illustration of the submucosal arterial blood supply to the lateral nasal wall; anterior and posterior ethmoidal artery branches are shown in outline form; sphenopalatine artery branches are shown in solid black.

The external carotid artery supplies the lower and posterior portions of the internal nose via a branch, the internal maxillary artery, from which are derived the major palatine artery and the sphenopalatine artery. The latter artery enters the internal nose at the rear through the sphenopalatine foramen. It supplies posterior lateral nasal branches to the middle and inferior turbinates and to their respective meati (the lateral nasal wall). A septal branch of the sphenopalatine artery supplies most of the surface of the septum, thus practically paralleling the arterial distribution on the lateral nasal wall (Figs. 1 and 2). The artery itself courses downward and forward along the nasal septum to the incisive (anterior palatine) foramen on the floor of the nasal cavity, where it anastomoses with the major palatine artery which enters the nose through that foramen. The distribution of the arterial branches from both of these systems on the septum practically parallels that on the lateral nasal wall and warrants reiteration for reasons which will be indicated later.

Evaluation of Types of Packing

Postnasal packing has been routinely applied to the general site of active posterior nasal hemorrhage in those cases in which cautery was not applicable and before vascular ligation would be considered. In conformity with the then contemporary thinking, Beinfeld[6] made the statement that it mattered little which kind of postnasal pack was used. He felt that they all act in the same manner by snugly closing off the posterior choanae and making pressure against the sphenopalatine artery on the floor of the sphenoid sinus and the septum.

Kuhn and Hallberg[6] rightly objected to the postnasal pack as previously routinely used for posterior intranasal bleeding since it essentially acted as a nasopharyngeal plug against the choanae and did not enter the nasal passages at all to contact the active bleeding points within them. They recommended a cone-shaped intranasal pack, the designing of which they credited to Dickie.[7] This was so constructed as to facilitate entrance into a nasal passage, via the posterior nares, by virtue of its tapering from the apex to the base. This pack has also been recommended by Woodruff.[4] Neivert[8] advocates the use of a nasal plug from a long dental roll to help combat posterior nasal bleeding without blocking the Eustachian tube. Saunders[8] describes an adaptation of a MED vaginal tampon for the control of postnasal hemorrhage.

There have been several types of inflatable balloon packs described. Cyril Fox has designed a postnasal pack which consists of a catheter 7 in. long assembled with a rubber bulb and a latex balloon. Johnson[9] describes an F-16 Foley balloon type catheter for postnasal bleeding. Both the Fox and the Foley balloon packs are more applicable to adenoid bleeding than to posterior intranasal hemorrhage. My own[10] experience with the Foley balloon catheter has not been too successful. Stevens[11] designed an improved version of his rubber pneumatic pack, which he finds has many advantages over the conventional types of packing.

Requirements for an Ideal Intranasal Pack

Most of the packs described are intended for unilateral use and are claimed to avoid such complications as blockage of the Eustachian tubes, laceration of the soft palate, hemotympanum, and pharyngeal edema. But being unilateral, they are kept in position by tension between the turbinates and the septum. They may, however, become dislodged from the source of hemorrhage by lessening of this tension due to shrinkage of the turbinal mucosa. Thus they do not satisfy the requirements as pronounced by Harkins,[12] who specifies that a properly placed postnasal tampon, with tension maintained, will promptly stop almost all bleeding in this area; while Beinfeld[6] states that a postnasal pack must be held securely in place if it is to be of any value.

Therefore, in the presence of an emergency situation in which there is a profuse hemorrhage from the posterior nasal passages and in which a posterior intranasal pack is indicated, the following conditions should be satisfied:

1. The pack should be placed intranasally via the posterior nares and not merely in the postnasal space.

2. It should be soft and pliable so as to mold itself easily around and within the contours of the turbinated bones and within their meati.

3. It should possess compressibility so as to permit insertion through a nasal passage narrowed by a deviated septum. This is the side more likely to harbor the bleeding source, since it is the convexity of the deflected septum which thins the mucous membrane, thereby exposing the submucosal vessels to trauma or stress.

4. It should be absorbent and, by virtue of this property, be capable of an increase in mass whereby compression against bleeding points would ensue.

5. It should exert equal pressure against the septum and the lateral nasal wall in order to exert simultaneous pressure against a septal or lateral nasal wall branch of the sphenopalatine artery which may be the source of hemorrhage. This double application of a single intranasal pack is feasible because of the practically parallel distribution of the septal branches to the lateral nasal wall branches.

6. The same single packing should be anchored at an immovable posterior fulcrum (Figs. 3 and

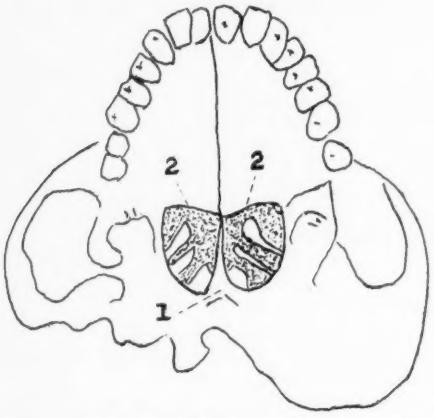


Fig. 3.—A view of the intranasal passages from the postnasal approach diagrammatically depicting the posterior border of the vomer (1) which is the central partition between the choanae (2).

4), the posterior border of the vomer (the central partition between the choanae) and at an immovable anterior fulcrum (Fig. 4), which is that area between the projecting anterior nasal spine and the anterior septal border.

7. The same single packing should be used bilaterally so as to be fixed and not be subject to displacement away from the source of hemorrhage.

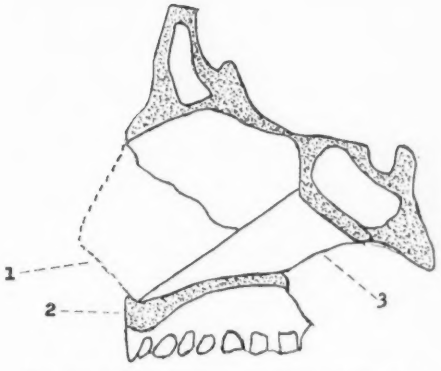


Fig. 4.—An outline of a lateral view of the septum showing the anterior fulcrum which consists of the area between the anterior septal border (1) and the anterior nasal spine (2), and the posterior fulcrum which consists of the posterior border of the vomer (3).

A Bilateral Posterior Intranasal Pack

A bilateral posterior intranasal pack has been developed according to the pattern shown in Figure 5.

A strip of gauze is folded in so as to eliminate the raw edges, producing a rectangle of gauze 3½ in. by 5½ in. In its center is attached the end of a 15 in. length of a ¼ in. wide umbilical tape. About ¼ in. on each side of this tape is placed a

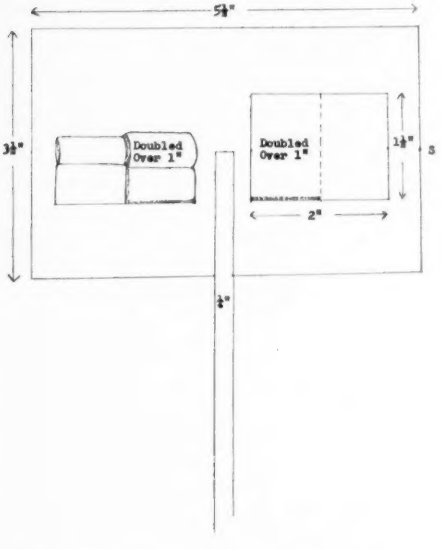


Fig. 5.—Schematic representation of the construction of the bilateral posterior intranasal pack.

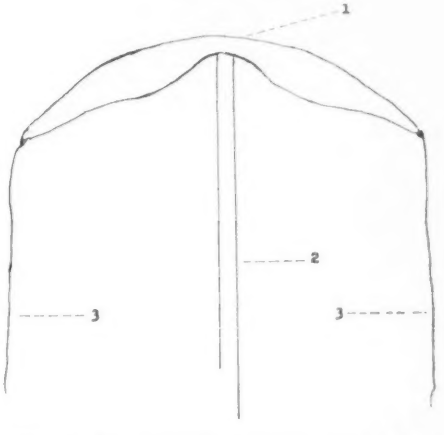


Fig. 6.—Diagrammatic representation of the bilateral posterior intranasal pack, ready for use, showing the "waist" (1) of the pack which is anchored behind the posterior border of the vomer, the tape (2) which hangs free of the soft palate, and the strings (3) which are anchored over the area between the anterior nasal spine and the anterior septal border.

1½ by 2 in. strip of compressed cotton which has been folded over itself by 1 in. (from an original 1½ by 3 in. strip) in order to taper it when rolled, producing the Dickie type of pack. The thicker end of each strip of compressed cotton is placed medially so as to produce the tapering effect when rolled tightly. The middle part, or "waist," of the pack is purposely left thin so as to permit flexibility when the pack is bent on itself when anchored posteriorly around the posterior border of the nose.

A string, 36 in. long, is stitched at the edge of the gauze (shown by the letter *S* in Fig. 5), leaving a 12 in. length of string hanging free. The stitching of the other end of the string is continued by sewing in the tape securely. After the gauze rectangle has been completely rolled tightly, several stitches are placed at the sides of the gauze packing in order to effect complete closure. Three or four stitches are now placed at one end to help produce a tapered effect, then back-tracked with a loop to the opposite end, which is also reinforced with stitches, leaving another 12 in. length of string hanging free (Fig. 6).

Method of Application

The method of application of this type of intranasal pack is similar to that used for the unilateral pack except that the bilateral approach is used. Two nasal catheters are passed simultaneously into the nasopharynx, one through each nasal passage. A string from each end of the above-described packing (the tapered ends of which have previously been lubricated) is now tied to the oral presenting end of each catheter. The latter are then withdrawn, each string respectively being pulled through each nasal passage. The two strings are grasped with one hand, and forward traction is exerted while, with the other hand holding a Mayo forceps, the pack is placed well up into the nasopharynx to facilitate its entry into the posterior nares. Further forward traction to each string will anchor the middle "waist" of the pack firmly against the posterior border of the vomer, which acts as the posterior fulcrum. The taut strings are then tied over a dental roll placed between the anterior nasal spine and the anterior septal border, which together act as the anterior fulcrum.

The ¼ in. tape now hangs loosely in the nasopharynx and may be cut 1 in. below the level of the soft palate so as to avoid traction caused by the act of swallowing. The use of this tape, instead of a string, precludes possible laceration of the soft palate.

Conclusion

The description of a procedure is usually more involved than its execution, and the construction and application of this pack is no exception to the rule. It is actually a relatively simple procedure.

This type of posterior intranasal packing is not a panacea for all types of intranasal hemorrhage. It is indicated in an emergency situation in which there is a massive intranasal hemorrhage from the posterior area and in which the source of bleeding cannot readily be determined. It is, similar to other nasal packs, merely a stop-gap procedure until the bleeding source can be localized when more definitive measures may be instituted.

The matrix of the packing contains compressed cotton which swells on absorbing blood and thereby exerts pressure on contiguous bleeding points. The use of a bilateral pack disposes of the immediate necessity of determining on which side the bleeder is located in emergency situations when the field is flooded with blood. Anchoring the pack both posteriorly and anteriorly renders it less liable to displacement. The size of this pack may be reduced proportionately in all of its dimensions in order to accommodate nasal passages smaller and narrower than the average adult size. Anterior packing with ½ or 1 in. gauze may be applied from the back of the nasal passage forward if reinforcement of hemostatic pressure is desired. The pack may be left in place for 24 to 48 hours and longer if necessary.

Summary

The practically parallel vascular distribution of the septum and of the lateral nasal wall is reviewed and emphasized.

The difference between postnasal and posterior intranasal packing is discussed with an evaluation of the various types of packing used.

The construction of a posterior intranasal pack, which meets specific requirements, is described.

Olive Whidden, R. N., Supervisor in Surgery, Hahnemann Hospital of San Francisco, assisted in the development and construction of the posterior intranasal pack.

516 Sutter St.

REFERENCES

1. Schaeffer, J. P.: The Nose, Paranasal Sinuses, Nasolacrimal Passageways, and Olfactory Organ in Man, Philadelphia, P. Blakiston's Sons & Co., 1920, pp. 275-278.

2. Davis, G. G.: Applied Anatomy, Philadelphia, J. B. Lippincott Co., 1910, p. 98.

3. Saunders, W. H.: Effective Control of Postnasal Hemorrhage, A. M. A. Arch. Otolaryng. 64: 291-292, 1956.

4. Woodruff, G. H.: Severe Epistaxis, A. M. A. Arch. Otolaryng. 67:435-439, 1958.

5. Beinfeld, H. H.: General Principles in Treatment of Nasal Hemorrhage, A. M. A. Arch. Otolaryng. 57:51-59, 1953.

6. Kuhn, A. J., and Hallberg, O. E.: Complications of Postnasal Packing for Epistaxis, A. M. A. Arch. Otolaryng. 62:62-65, 1955.

7. Dickie, J. K. M.: Nasal Hemorrhage and Its Treatment, Canad. M. A. J. 58:121-126, 1948.

8. Neivert, H.: Principles and Mechanics of Surgery of the Bony Nasal Vault, A. M. A. Arch. Otolaryng. 61:585-593, 1955.

9. Johnson, F.: The Control of Adenoid Hemorrhage with a Foley Catheter, A. M. A. Arch. Otolaryng. 63:295, 1956.

10. Gidoll, S. H.: Postoperative Nasal Hemorrhage, A. M. A. Arch. Otolaryng. 67:530-535, 1958.

11. Stevens, R. W.: Nasal Packing: The Rubber Pneumatic Pack, A. M. A. Arch. Otolaryng. 54:191-194, 1951.

12. Harkins, H. P.: Hemorrhage in Nose and Throat, A. M. A. Arch. Otolaryng. 55:8-11, 1952.

New Instrument—Roller Knives

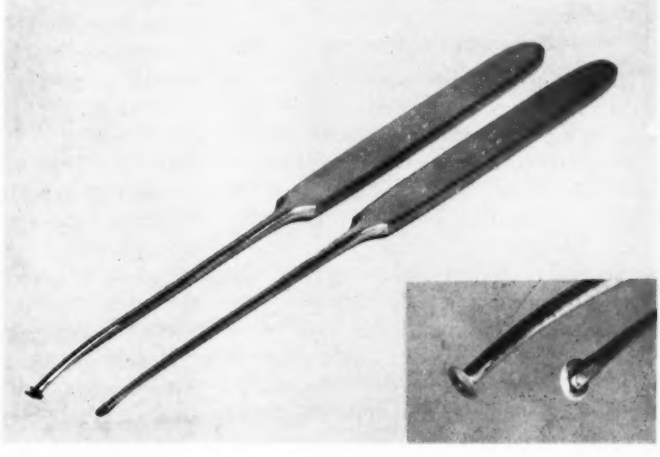
HAROLD F. SCHUKNECHT, M.D., Detroit

The instruments * described here are designed to incise the skin of the bony external auditory meatus when creating the tympanomeatal flap for transtympanic surgical procedures. The wheels are 3 mm. in diameter, have a sharp cutting margin, and when they are rolled firmly against the bony wall, they cut through the skin and periosteum without tearing the skin. The wheel should be rolled two or three times in the same line of incision. One wheel is parallel and the other at right angles to the shaft. Ordinarily 50 to 100 operations can be performed with a set of knives without sharpening.

Submitted for publication Aug. 20, 1958.

* Available from V. Mueller & Company, Chicago.

Henry Ford Hospital (2).



Roller knives

Progress in Otolaryngology

Bronchoesophagology

Summaries of the Bibliographic Material Available for 1957

F. JOHNSON PUTNEY, M.D., Philadelphia

In 1957 bronchoscopy was used chiefly in the diagnosis of bronchogenic carcinoma and in the treatment of postsurgical complications. Esophagoscopy became firmly established in the diagnosis of esophageal disease with or without positive roentgen findings, as evidenced by its increasing use in hiatal hernia. Fundamental studies of pulmonary function, esophageal motility, and esophagogastric sphincteric mechanism received more attention, and basic research in the latter helped to clarify some of the controversial points regarding the hiatal sphincter.

Experimental Studies

Hilding [1] studied the ciliary activity of the lower respiratory tract in humans and animals. Ciliary activity was found to continue many hours after death in humans, while the mucous blanket behaved less dependably. The flow of the ciliary stream in the bronchial tree of man was found to be generally upward against gravity and was very wide but rapidly narrowed as it approached the trachea. The velocity of ciliary flow was estimated as an average 14 or 15 mm. per minute. A substantial part of the visible smoke from ordinary cigarettes was found deposited in the lower respiratory tract upon the mucous blanket wherever the inspired air flowed. Part of the smoke was dissolved and absorbed and the rest was carried away with the mucous blanket in the ciliary stream to the esophagus. Areas of slowing or comparative stasis of the ciliary action and movement of the mucous blanket were found in various portions of the tracheobronchial tree and this was considered a possible depot for carcinogenic substances. The islands of squamous epithelium occurred where the ciliary stream narrowed and obstructions were met, so that any inhaled slowly acting carcinogenic substance might be expected to accumulate in these areas and exert more activity.

Dekker and Groen [2] submitted lungs to different pressures in a closed jar and found an expiratory pressure of 1 cm. of mercury or more produced considerable stenosis by prolapse of the membranous trachea and main bronchi into the lumen. A similar passive compression of the larger air passages caused by the high intrathoracic expiratory pressure was considered an important factor in the production of asthmatic bronchostenosis and expiratory wheezing.

Deloyers and his co-workers [3] were able to produce experimental cardiospasm in cats by injection of 5% phenic acid between the two coats of smooth muscle in the wall of the cardia. The disease produced was similar to that of humans clinically, radiologically, and anatomically. The microscopic lesions were essentially destruction of the ganglion cells of Auerbach's plexus.

Muren [4] established an esophageal fistula in dogs which retained its sphincter function. After the esophagus was divided in the neck, the lower end was exteriorized and the upper end was anastomosed to the side of the lower portion. The fistula thus formed closed automatically when swallowed food passed the region, and functioned satisfactorily for periods of a year or longer.

Submitted for publication Oct. 6, 1958.

The sphincteric mechanism was apparently due to the arrangement of the esophageal propulsive activity along the entire organ which was retained after interruption and displacement of the muscular tube provided the nerves were not severed.

Peroral Endoscopy. Photography and Television

Jackson [5] described some of the mechanical changes in the endoscopic instrumentarium. Removal of the rim at the distal end of the bronchoscope and flaring the proximal end of the tube proved advantageous. Esophagoscopes were prepared in the same manner, but both round and oval lumens were designed. The handle on the classic laryngoscope was revised to give a firmer grip also. The "fotobronchoscope" utilized the quartz rod illuminated principle similar to that used by Forestier in his photographic bronchoscope.

Holinger and Johnston [6] discussed the newer phases of bronchoscopy in diagnosis and treatment, including endoscopic photography. The bronchoscope has been used in catheterization of the left heart by transbronchial puncture, in which a polyethylene tube is passed through a needle to the left ventricle and pressures on the two sides of the mitral valve are recorded by separate manometers. After extensive thoracic surgical procedures bronchoscopic problems of freeing secretions and granulations from the areas of anastomosis and skin grafts have arisen. Bronchoscopic replacement and splinting of the bronchial walls has assisted in the management of fractures of the bronchus. Bronchoscopy in the newborn aids in differentiation of atelectasis from agenesis, endobronchial webs from extrabronchial compression as a cause of obstructive emphysema, and other congenital anomalies. The bronchoscopic aspects of pulmonary suppuration in fibrocystic disease of the pancreas were discussed. Not only is the field of bronchoscopy changing, but the need for bronchoscopy in both diagnosis and therapy is increasing, and the requirements listed for safe and effective bronchoscopic examinations were manual dexterity, training in monocular vision procedures, and constant practice.

Using a new type of bronchoscope with a proximal light source, telescope and a quartz rod to transmit the light to the distal end of the instrument, sufficient illumination was obtained by Soulas and his associates [7] to permit attaching a television camera directly to the endoscope. The picture was transmitted to screens in the operating rooms and in various locations in the hospital through a closed circuit and was thought to be of great value for both teaching and consultation. The bronchoscopic pictures of several patients were transmitted simultaneously on several screens, and students and physicians grouped around the screen were able to observe at the same time as did the bronchoscopist a tuberculous stenosis, a neoplastic stenosis, and the alteration of the bronchi in bronchiectasis. The second application of this method was on an anesthetized dog and permitted the viewers to observe spectacular bronchial spasm resulting from excitation of the vagus nerve.

The changing concepts of bronchoesophagology were discussed by Cracovaner.[8] Not only has the armamentarium improved and endoscopic photography been developed, but the influence of bronchoesophagology has extended to other specialties. The type of case formerly seen by the bronchoesophagologist has changed in that very few cases of lung abscess, bronchiectasis, and tuberculosis are now observed. Bronchoscopy is still useful in tuberculosis but its most important field remains in the diagnosis of cancer of the lung.

Som and Arnold [9] discussed esophagoscopy in the diagnosis and treatment of esophageal disease and reviewed the endoscopic findings in various conditions.

Anesthesia

In discussing anesthesia for bronchoscopy Wyant and his associates [10] concluded that local anesthesia was the safest method and should be routinely employed. General anesthesia was used routinely for bronchoscopy

in children and in those cases not satisfactorily managed under topical anesthesia. A method of balanced analgesia, sedation, and hypnosis combined with relaxation and local anesthesia was described for those cases unsuitable for local anesthesia alone. Sedation was induced by Thorazine and small amounts of Demerol were used in conjunction with Nalline for analgesia. Topical anesthesia was administered while the patient was sedated and relaxed.

Hughes et al.[11] evaluated Dyclone (dyclonine) and Cyclaine (hexylcaine) as anesthetic agents for endoscopic procedures. In dogs intravenous Dyclone was less toxic than Cyclaine, tetracaine, or cocaine, while Cyclaine was slightly less toxic than tetracaine and cocaine but the difference in the latter was not great. Clinically mild reactions were less frequent with these agents than they were with tetracaine and cocaine and both Cyclaine and Dyclone produced adequate anesthesia in most patients for 30 to 40 minutes. This study was carried out on 665 patients and both anesthetic agents proved satisfactory with respect to potency, promptness of action, and freedom from untoward reactions.

Coughlin and his associates[12] believed that better topical anesthesia of the lower respiratory tract was obtained by transtracheal administration than by other methods and recommended its use for reflex inhibition in bronchoscopy performed either awake or asleep. With a 20 or 21 gauge needle 2 cc. of various anesthetic agents were injected into the airway through the cricothyroid membrane in the midline.

Gonzalo[13] reported the use of general anesthesia together with artificial hibernation in a series of patients undergoing examinations of the air and food passageways. In the Latin countries it was necessary to use some form of general anesthesia because of the make-up of the patients so examined. The method employed reduced the amount of anesthetic to a minimum, avoided shock and side-reactions and simplified the induction of anesthesia. Anesthetization by artificial hibernation was employed in patients who were frightened, did not cooperate or relax, and in those in which previous local anesthesia had been unsatisfactory. It was also used in patients with local pain, in older children, in cases of foreign bodies in the esophagus, in cases in which prolonged intervention was necessary, in those with a history of toxic reaction to local anesthetic, in suspension laryngoscopy, and in very old patients that were increased surgical risks. The method consisted of administering pentobarbital 0.1 or 0.2 gm., depending on the body weight the evening before the examination, and the intramuscular injection of 50 mg. of promethazine. One and a half hours before the examination an intramuscular injection consisting of promethazine 50 mg., diethazine 250 mg. and meperidine .01 mg. was administered. One-half hour before operation an intravenous infusion consisting of 500 cc. of dextrose in water to which had been added 1 cc. of thiamine, 50 mg. of promethazine, 100 mg. of meperidine and 50 mg. of chlorpromazine was administered at the rate of 30 to 50 drops a minute. A few minutes before the patient entered the operating room the pharynx was sprayed with tetracaine 2% or cocaine 10%.

Intravenous meperidine in doses from 100 to 200 mg. was used as an analgesic agent in peroral endoscopies in conjunction with local anesthesia in a series of 250 cases by Altman and Fialkov.[14] The advantages noted were muscular and psychic relaxation with reduction of the amount of local anesthetic agent required and the absence of reaction. The method of anesthesia consisted of 1 mg. of atropine sulfate hypodermically one hour prior to endoscopy, followed by the instillation of 0.5% tetracaine solution in amounts not exceeding 8 cc. or 5% cocaine solution in amounts of not more than 5 cc. into the larynx prior to the examination. Meperidine was administered intravenously slowly with the patient on the operating table, a 1% solution being used.

Johnston and his associates[15] reported a tetracaine reaction in an 85-year-old man followed by cardiac arrest and resuscitation.

After preliminary medication with morphine and scopolamine both 5% cocaine and 2% tetracaine were used to anesthetize the pharynx and larynx, an estimated 5 cc. of the latter medication being used. Pentobarbital sodium, 2 grains (0.12 gm.), was given intravenously followed by 30 mg. of gallamine triethiodide. Five minutes after the last application of the anesthetic the pulse suddenly stopped although no instrumentation had been done before this happened. Thoracotomy was performed, and the heart resumed its normal beat with no permanent after-effects.

Congenital Anomalies

Holinger and Johnston [16] pointed out that bronchoscopy without anesthesia should be done as a diagnostic procedure in any unusual respiratory condition in infants. Limiting the time of the procedure, the use of small instruments and the administration of oxygen through the bronchoscope during the examination increased the margin of safety. The diagnosis of congenital malformations of the lower respiratory tract during life was made with the help of various specialists, including the pediatrician, roentgenologist, bronchologist, and thoracic surgeon. Anomalies of the trachea included constriction and enlargement, evagination, outgrowth, and abnormal bifurcation or deviation. Anomalies of the bronchi and lungs were described as agenesis, constrictions, evaginations, subnumerary and supernumerary bronchi, lobes and fissures and anomalous bronchial and lung tissue attached to the respiratory or other systems. Acute respiratory symptoms in the newborn period required prompt diagnostic studies, particularly those caused by various degrees of bronchial obstruction due to cardiac and vascular malformation.

A case of tracheal atresia associated with tracheoesophageal fistula was presented by Devenis and Otis.[17] Respirations were difficult at birth and tracheal intubation was attempted but the tracheal tube could not be inserted. After death the diagnosis was made by the autopsy finding of complete closure of the trachea just below the vocal cords. There was a tracheoesophageal fistula just above the carina, and through this opening the child received all of its respiratory air.

A rare type of tracheoesophageal fistula occurring at the level of the cricoid cartilage without atresia was reported by Kraus and White.[18]

Babbitt [19] reported an unusual case of a double tracheoesophageal fistula without atresia. Death occurred because only one fistula was recognized and repaired surgically. The repaired fistula proved to be the lowermost one, for at autopsy a second fistula 3 mm. in diameter was found in the suprasternal region at the upper end of the trachea just below the larynx and communicating with the upper esophagus.

Shaw and Paulson [20] reported their salvage rate for atresia of the esophagus corrected surgically in 65 babies as 41.5%, with improvement during the past five years to 65%.

The anomaly of a bronchus arising from the right side of the trachea was reported by Schaff and Baum [21] in two patients as an example of a displaced apical segment.

Nelson [22] reported an unusual case of a congenital esophageal diverticulum in a 2-year-old child at the cricopharyngeus muscle unassociated with other congenital abnormalities. Attention was called to the condition by the lodgment of a foreign body (penny) in the cervical pouch. After esophagoscopic removal the lesion was demonstrated. Partial excision of the sac was carried out, and histologic section revealed all of the coats of the esophagus to be present. It was necessary to treat the residual lesion after surgery by forceful endoscopic dilatation to secure a satisfactory functional result.

A case of intramural duplication of a segment of the thoracic esophagus causing dysphagia first in adult life was reported by Maier.[23] The correct diagnosis of a double-barreled lumen with one channel having a blind lower end was made

roentgenologically only after esophagoscopic manipulation and biopsy of a web.

Agenesis of Lung

A case of agenesis of the lung was reported by Netterville[24] with all of the characteristic clinical findings. On bronchoscopy the carina was much wider than normal and the right main bronchus ended abruptly about 1 cm. beyond the bifurcation of the trachea. This 6½-month-old youngster was followed for five years and was extremely active with no dyspnea even on extreme exertion so that the prognosis appeared excellent.

Sullivan[25] reported a case of agenesis of the right upper lobe which on bronchoscopy revealed only slight indentation where the right upper lobe normally should be. The mediastinum was displaced to the right and iodized oil studies revealed only a small stump of the right upper lobe bronchus.

Nicks[26] reported a case of agenesis of the lung with persistent ductus arteriosus. At bronchoscopy the trachea was normal but the carina was distorted marking the origin of the left main bronchus which was about half the diameter of the right and lined by healthy mucosa. The left main bronchus terminated 1 in. from its origin in a blind pouch. These findings were confirmed by bronchography.

Diverticulosis of Trachea

A case of tracheal diverticulosis was described by Goldman and Wilson.[27] Roentgen studies revealed a radio-opaque object parallel to and between the trachea and the cervical spine at the level of the sixth cervical vertebra. Immediately below this area there was some radiolucency and the trachea was widened in its midportion. The esophagus was deviated in a smooth curve but no esophageal diverticulum was found. A foreign body which proved to be a piece of bone was removed, and subsequent tracheograms using radio-opaque material revealed a diverticulum of the trachea with a relatively narrow inlet and numerous other smaller diverticula located on the right posterior aspect of the trachea and varying from 3 mm. in diameter to 2×1 cm.

Rupture of Trachea

A case of complete traumatic rupture of the cervical trachea following a closed chest injury (steering post) was presented by Beskin.[28] Primary anastomosis of the severed trachea and distal tracheostomy were used with excellent results.

Tracheal Stenosis

The management of tracheal stenosis was discussed by Cummins and Waterman[29] who reported a case of a young woman with almost complete stricture of the trachea 6 cm. in length just above the carina and secondary to a severe chest injury. Bronchoscopy performed on several occasions revealed marked granulations throughout the trachea which responded temporarily to antibiotic therapy. Even though the granulations disappeared smooth stricture formation soon followed, so surgical treatment was undertaken with resection of the stricture and subsequent replacement by a skin graft reinforced by tantalum gauze.

Tumors of Trachea

Greer and Winn[30] reported a case of a leiomyoma of the trachea diagnosed by bronchoscopy. The patient had symptoms of asthma, and bronchoscopy revealed a pink, smooth, slightly lobulated tumor in the distal trachea almost occluding the entire lumen just above the bifurcation. There was only a small air passage to both main-stem bronchi anterior to the tumor, and tissue removed was reported benign. Piecemeal removal of the tumor was performed through the bronchoscope and about one-third of the lesion was destroyed so that an airway was established. After removal of a portion of tumor it was evident that it arose from the membranous portion of the trachea in an area about 1.5 cm. in diameter, so excision at subsequent thoracotomy was necessary.

Gibbs and his associates [31] reported the first case of von Recklinghausen's disease in the trachea. Plexiform neuroma in a 16-month-old infant produced respiratory obstruction by involvement of the larynx and circumferential extension around the trachea. The tumor did not involve the overlying mucosa or invade cartilage when examined at necropsy.

Barkley and his associates [32] reported a case of cylindroma of the trachea involving the carina and the origin of the right and left main bronchi, in which the tumor was resected and the continuity of the bronchial tree was restored without the use of grafts throughout a 5 cm. gap in length. Using the technique of mobilization of the lower end of the trachea it was found possible to resect 6 cm. of trachea including the carina if necessary and reconstruct the bronchial tree without the use of grafts.

Acquired Esophago-Respiratory Fistula

Coleman [33] reviewed 93 reported cases of acquired nonmalignant esophago-respiratory fistula. The causes were trauma, infection, and diverticulum of the esophagus. Trauma proved to be the most frequent cause, and a correlation was found between the type of trauma and location of the fistulous orifice in the respiratory tree. The commonest specific infections leading to fistulous communications were tuberculosis and syphilis, while traction diverticuli of the esophagus revealed a high instance of communication with the secondary bronchi. The symptoms were characterized by ingestion of fluid followed by strangling or severe coughing after a momentary asymptomatic pause. The diagnosis was based upon the history, physical examination, roentgenographic studies, and endoscopic examination. Endoscopic examinations of both the esophagus and tracheobronchial tree were needed in some cases, and the area of traumatic disruption was identified in the immediate post-traumatic period by this examination. In patients with long-standing fistulas endoscopic examination revealed the fistula usually lying in the anterior lateral wall of the esophagus, and it was often overlooked owing to its slit-like configuration. Biopsy was used in papillomatous or ulcerating lesions, while bronchoscopy proved to be of value in confirming the communication of the fistulous tract with the respiratory tree when the fistula was in the membranous wall of the trachea or bronchus. Patients with small fistulas associated with large granulations were treated by local cauterization and antibiotics. Those with active tuberculosis or syphilis received adequate and appropriate therapy, while the other patients were treated by pulmonary surgical procedures.

Bronchography

One hundred twenty-four bronchograms were performed in a hundred children by Bovornkitti and Zabriskie,[34] with 110 satisfactory results. Successful bronchograms in children depended upon anesthesia, choice of contrast material, careful positioning of the patient to insure complete filling of all segmental bronchi, and the radiographic technique. Anesthesia was secured by the administration of scopolamine followed by open-drop ether, and a catheter was inserted into the tracheobronchial tree by direct laryngoscopy. Three contrast media, i. e., Lipiodol, Dionosil, and Visciodol, were used and the best results were obtained with Visciodol. The material was warmed and stirred vigorously before instillation, and better than 85% satisfactory bronchograms were obtained. Untoward reactions were noted and cyanosis occurred if both lungs were filled simultaneously with the opaque solution. Aspiration of the first side before injection of the second side was recommended. High iodine levels and enlargement of the thyroid following the use of both Lipiodol and Visciodol were observed as minor complications.

Bronchography was carried out with Dionosil oily in rabbits by Björk and Lodin.[35] After three months residual oil was found in 8 out of 10 animals and after six months in 7 out of 10 animals. Foreign-

body granulomata were more numerous in the latter group. Abandonment of the oily solution in favor of the aqueous suspension was advocated.

Visciodol was used as the contrast medium for bronchography in 35 cases by Pesiri and Sennott.[36] With this agent excellent bronchograms were obtained and practically never did the solution penetrate the alveoli. Since the material contains Lipiodol with sodium sulfite and sulfanilamide, a history of sulfonamide sensitivity should be inquired into prior to administration of the drug.

Haidak and his associates[37] reported a method of utilizing air injection into the tracheobronchial tree after filling all the segmental bronchi with a water-soluble radio-opaque substance (Dionosil). Between 150 and 200 cc. of air was injected quickly after all of the segments were filled with the contrast material so that the radio-opaque agent adhered to the bronchial mucosa and produced contrast mucosagrams.

Copp and his associates[38] performed 602 bronchographic studies on 400 consecutive patients with pulmonary tuberculosis and found an over-all incidence of bronchiectasis in 43%.

Bronchospirometry

Autio[39] reported his experience with 296 bronchospirometric examinations, of which 272 were successful, and concluded that the investigation is not simple and requires an experienced team for accurate results. The Carlens catheter was used, no serious complications were encountered, and no deterioration or spreading of the tuberculous process was noted under preoperative chemotherapy. The bronchospirometric total vital capacity was on the average 4.6% lower than the spirometric vital capacity, the bronchospirometrically measured ventilation on the average 23.8% greater than the spirometric value, and the oxygen uptake 5.3% greater during bronchospirometry than during spirometry. Sore throat, transient cough, hoarsenesss, and temporary slight temperature elevations were the only complications observed. The radioplanimetric method of examination was used advantageously to evaluate differential lung function and was partly substituted for bronchospirometry, making it possible to select and determine better than previously the patients upon whom bronchospirometry was definitely indicated.

Swenson and Birath[40] found that the Carlens catheters caused less resistance to air flow than other types. Small differences in tube design assumed considerable importance in clinical evaluation of comparative unilateral lung ventilation studies.

Birath and his co-workers[41] studied a series of 51 patients with pulmonary tuberculosis before and after segmental resection or lobectomy for unilateral functional loss. The loss of function three months postoperatively was larger than the amount of resected tissue with the greatest deviations between physiologic and anatomic loss occurring in those with the greatest restriction of ventilatory movements. Functional improvement after the three-month postoperative period was predicted.

A series of 17 patients with pulmonary resections were studied by Birath et al.[42] by bronchospirometry. The definitive functional loss for the smaller resections was only slightly larger than the calculated anatomic loss, while in the more extensive resections the final functional loss was larger than the number of segments would indicate.

Twenty-two patients undergoing thoracoplasty whose opposite lungs were apparently healthy were studied bronchospirometrically by Birath and Soderholm[43] before operation and three months postoperatively. The greater part of the functional decrement was found to be due to the disease, and after stabilization it was estimated that the total functional loss for the diseased and surgically treated lung was about one-third its normal value.

A method for the study of individual lung function during thoracic surgical procedures utilizing a double-lumen endotracheal bronchial tube was presented by T. R. Watson Jr. et al.[44] In the bronchospirometries an average shift of 4% in oxygen uptake of the

individual lung and 7% in ventilation was found. Usually greater participation in the total share of the ventilatory work and oxygen uptake was performed by the dependent lung in the lateral decubitus position.

Bronchospirometry during exercise was performed by Viikari and Autio[45] on 21 patients with pulmonary tuberculosis. The examination was carried out in the sitting position with no noxious effect on the patients and indicated that exercise did not change the relation between the oxygen intake of the two lungs. Cases of thickened pleura with favorable values for ventilatory equivalents during exercise were considered good candidates for lung decortication due to the adaptability of the blood circulation.

Oxygen Saturation During Bronchoscopy

DeKornfeld and Siebecker[46] studied the oxygen saturation in 50 patients during bronchoscopy for pulmonary disease and found the oxygen saturation rose in all patients.

Atelectasis

Eight cases of massive atelectasis of the left lung in infants with congenital heart disease were described by Rivkin and his co-workers.[47] The etiology was considered to be compression of the left main bronchus by the left pulmonary artery.

Mucoid impaction of the bronchi occurring in 36 patients, most of whom were asthmatics, was described by Shaw and his co-workers.[48] Bronchoscopy and bronchography both proved useful in diagnosis. Since the condition was present in the upper lobes in two-thirds of the patients bronchoscopy did not allow visualization in many. Not only was the detection of impacted mucus in the lower lobes possible, but removal by suction or forceps was often accomplished. The main diagnostic value of bronchoscopy was to exclude neoplasm.

Foreign Bodies in the Bronchus

Sixteen cases of chronic intrabronchial foreign body were reported by Linton.[49] There was no history to suggest the original instant, and the clinical picture was usually confused by other pathologic changes such as atelectasis, pneumonitis, bronchiectasis, or lung abscess, while the bronchoscopic appearances were often indefinite. When granulation tissue was observed on bronchoscopic examination and no foreign body could be seen it was advisable to rebronchoscope the patient and in many instances remove granulations before the foreign body could be visualized. The appearance of heaped-up friable granulation tissue should have suggested the presence of a foreign body but in five cases the clinical diagnosis by the bronchoscopist was carcinoma. The bronchoscopic appearance of carcinoma with the biopsy showing inflammatory changes should suggest the diagnosis of foreign body even though the history has been negative.

Semple and Page[50] reported a case of bilateral bronchial foreign body. A metallic radio-opaque reed from a toy whistle was removed from the right lower lobe bronchus about four hours after its inhalation, but the plastic mouth piece remained in the left lower lobe bronchus for 10 months and was not recognized until a fourth bronchoscopy, when it was removed. The latter tubular plastic foreign body produced no localizing radiological signs, but only reflex bronchospasm was noted.

DeRoos[51] reported a case of an adult with a persistent cough for three months which gradually developed into tracheal obstruction without a certain history of foreign body aspiration. Cure was effected by removal of a fairly large piece of dental plate from the right main bronchus which had been present for about 18 months.

Endobronchial Tuberculosis

Bronchoscopic studies in pulmonary tuberculosis in a series of over 500 cases were made by Palva and his co-workers.[52] Routine bronchoscopy was performed on each patient before any collapse or operative procedure and about one-half of the cases

fell into this category. Bronchial evaluation of the activity of the disease and differential diagnosis formed the second largest group. Other indications such as hemoptysis or atelectasis made up only a small minority. Anesthesia was induced after barbiturate, atropine, and morphine sedation using less than 10 cc. of 0.25% to 0.5% Pontocaine. The latter solution was used in fractionated doses, and no toxic reaction occurred. Changes indicating bronchial tuberculosis were present in about 10% of the cases. Granulations and ulcerations of the mucous membrane with fibrostenosis and fistulae were specific changes observed, while hyperemia and edema were considered nonspecific changes. Whenever possible the diagnosis of tuberculous bronchitis was confirmed by biopsy. The result of microscopic examination was almost always consistent with the clinical judgment, while biopsies taken in 19 cases with various degrees of hyperemia and slight edema revealed no tuberculous change. The fear of permanent ulceration following biopsy was not borne out, for repeated bronchoscopies in many cases after an interval of a few weeks failed to reveal any sign of irritation due to the previous biopsy. Endobronchial tuberculosis manifested itself in the form of granulations often associated with severe bronchitis, but generally there was no clear macroscopic tuberculous ulceration of the mucous membrane. Since bronchoscopic observation is limited to the main and lobar bronchi with the segmental orifices, bronchography was performed and gave invaluable information on the condition of the medium-sized and small bronchi. With the aid of bronchograms tuberculous bronchitis was discovered in nearly 50% of the cases. Bronchography was performed through the bronchoscope with an iodized oil-sulfonamide mixture which had the advantage of not entering the alveoli. Bacterial and cytologic studies were used to indicate the severity of the infection and the condition of the ciliated epithelium.

The indications, method, and interpretation of the findings at bronchoscopy in tuberculosis were discussed by Hulse.[53] The indications were listed as wheeze, blocked cavity, tension cavity, source of positive sputum, persistently positive sputum, low vital capacity, blood streaking, postresectional inspection, intractable cough, atelectasis, and suspected malignancy. Local anesthesia was employed using 1% tetracaine in the pharynx and larynx after premedication with pentobarbital, morphine, and atropine. On a few cases tripelennamine 2% was used for anesthesia and produced a bitter taste, increased salivation, distress in breathing and less ease in introduction of the bronchoscope, so it was discontinued. The forms of endobronchial tuberculosis observed were superficial lesions, submucosal lesions, sinus tracts, stricture, narrowing, and kinking.

Endobronchial lesions in pulmonary tuberculosis were considered essentially obstructive, and the limitations of antituberculous chemotherapy were emphasized by O'Keefe.[54] Bronchoscopy was considered indispensable in the diagnosis of the tuberculous patient, and repeated endoscopic examinations were necessary to uncover the complication of tuberculous bronchitis. While bronchoscopic examination determined the presence of endobronchial lesions and the type, location, and extent of involvement, it also afforded a means of assessing the progress of treatment, contraindication to surgical therapy, and direct therapy to the lesions when indicated. In lymph-node tuberculosis there was observed extraluminal compression of the tracheal or bronchial walls progressing to actual invasion of the walls by the tuberculous nodes with edema, polyp formation, ulceration, granulation, and even perforation with intrusion of caseous material into the lumen.

Grosz[55] reported a case study in which there were three perforations of tuberculous lymph nodes into different bronchi of the same lung. Early in the course of the tuberculosis a large complex of tuberculous lymph nodes perforated into the right upper lobe bronchus, causing destruction of the bronchial wall and distally an aspiration

infiltration of the lung. A year later relapse coincided with perforations of tuberculous lymph nodes into the apical bronchus of the lower lobe, which led to the development of a cavity. At operation a scar in the main bronchus of the lower lobe was observed and considered also to have been a result of lymph node perforation.

Sarcoidosis

Eleven consecutive cases of pulmonary sarcoidosis were investigated by Kalbian[56] by bronchoscopy, bronchial biopsy, and scalene node biopsy. Four of the eleven cases had no specific bronchoscopic abnormality except slight increase of mucus secretion and a tendency to bleed easily from the mucosa. Two cases revealed distortion of the carina presumably due to enlarged mediastinal lymph nodes which were evident on the roentgenogram. In four other cases which also showed distortion of the carina the mucosa was granular and rough looking and bled easily. The granulations were small bleb-like elevations 2 to 3 mm. in diameter. Two cases had very thickened mucous membrane with narrowing of the bronchus and edema while in one case the mucosa was acutely inflamed. This investigation suggested that the bronchi are frequently involved in pulmonary sarcoidosis and the diagnosis can usually be determined by histologic study of bronchial biopsy material. A combination of bronchial biopsy and scalene node biopsy was considered the best way to obtain histologic evidence of pulmonary sarcoidosis. The three abnormal bronchoscopic findings in pulmonary sarcoidosis were (1) external pressure from enlarged lymph nodes; (2) granular, nodular, rough mucous membrane with small blebs, and (3) thickened edematous mucous membrane with stenosis of the bronchus.

In three patients Citron and Scadding[57] observed stenosis of the main and segmental bronchi due to granulomatous changes in the walls from sarcoidosis. Bronchoscopic examination revealed narrowing of the bronchi with thickening of the mucosa from which tissue was removed for histologic study. The tissue reports aided little in the diagnosis, but bronchographic study proved valuable in determining the degree and extensiveness of the narrowing.

The occurrence of bronchial lesions in sarcoidosis was demonstrated by two cases reported by Honey and Jepson.[58] The clinical features were recurrent bronchopulmonary infection, stridor, and progressive dyspnea with no extrathoracic manifestations of sarcoidosis. Bronchoscopy revealed multiple bronchial stenoses, and tissue was reported as discrete, noncaseating epithelioid follicles, thereby establishing the diagnosis. Treatment consisted of antituberculous drugs and cortisone.

Fungus Infections of Lungs

A case of primary bronchopulmonary aspergillosis was reported by Stevenson and Reid.[59] On bronchoscopy large amounts of purulent secretions were observed arising from a lung abscess with congestion of the mucosa of the left main bronchus. Tissue removed from this area revealed only severe nonspecific inflammation. Aspergillus was recovered from the culture of the pus. Temporary improvement followed hydroxystilbamidine therapy.

Polk and his associates[60] considered pulmonary resection to be the treatment of choice in chronic progressive pulmonary histoplasmosis, although the surgical follow-ups have not been sufficiently long to completely evaluate this method of treatment. Since pulmonary histoplasmosis and tuberculosis occur together frequently, antituberculous therapy was used prior to resection. Reliance was also placed on the histoplasmin skin test for diagnosis.

Benign Tumors of Lung

Two cases of asymptomatic benign granular-cell myoblastoma of the bronchus were reported by Peterson and his associates.[61] In one case the diagnosis was made by bronchoscopic examination, which revealed a ridge in the right upper lobe

bronchus that obscured but did not occlude the orifice to the anterior segmental bronchus. The surrounding bronchial mucosa was red and appeared chronically inflamed, and tissue removed from the ridge-like area was reported as granular-cell myoblastoma. The remainder of the tumor was removed by endobronchial manipulation one week later. In the other case the bronchoscopic findings were normal, and the lesion was removed at operation. The tumor does not undergo malignant change and often presents as a polypoid sessile mass causing various degrees of bronchial obstruction with resulting pneumonitis. Infiltration through the bronchial wall may occur so that endoscopic removal must be complete to insure a cure. Careful follow-up of the patient after endoscopic removal is necessary since the neoplasm may recur if incompletely excised.

A case of lipoma of the bronchus removed endoscopically was reported by Ochsner and his associates.[62] Bronchoscopy revealed a smoothly rounded firm tumor which almost filled the right bronchus and when removed measured 3 cm. long and 5 mm. in diameter. Because of the persistence of bronchiectasis below the obstructing tumor mass surgical resection of the right middle lobe was necessary.

Adenoma of Bronchus

Fraser[63] published accounts of 12 cases of carcinoid type of bronchial adenoma. In eight cases the correct diagnosis was reached by bronchoscopy on the initial examination, while in one case further preoperative bronchoscopy was necessary; in one case the diagnosis was only made by examination of the postoperative specimen, and in one case the true nature was established three years later by further study of the histology. In one of the cases the lesion was successfully removed through the bronchoscope, but subsequent lobectomy was required to remove diseased lung tissue.

Overholt and his associates[64] reported 60 patients with bronchial adenoma undergoing pulmonary resection. The correct diagnosis was established by bronchoscopy in 25 cases in this series, but biopsy was not routinely done because of the fear of serious hemorrhage. In 67% of the cases invasion of the bronchial wall was present at operation, and approximately 15% had lymph-node and distant metastasis. Six of the patients had been treated by bronchoscopic resection with local recurrence of the tumor in each instance. Four patients had multicentric lesions. In the 20 cases operated upon more than 10 years before all were well except one case which died from the tumor.

Ochsner and Ochsner[65] reported 16 cases of bronchial adenoma, and in the 13 cases in which bronchoscopy was done positive findings were obtained in 12. Biopsy was performed in nine cases, with seven diagnosed as adenoma, one called carcinoma, and one containing no tumor tissue. Profuse postbiopsy bleeding developed in two cases. Most of these tumors arose in the larger bronchi and were directly accessible to bronchoscopy. In treatment bronchoscopic removal was incomplete unless the tumor was attached by a small stalk; recurrence of the tumor was common; the permanent disease beyond the tumor was not relieved by this means, and the chances of distant metastasis were greater.

Thompson and his co-workers[66] reported a case of bronchial adenoma with metastasis to the regional lymph nodes.

Carcinoma of Lung

A systematic examination of the tracheobronchial trees of 54 white men who died of bronchogenic carcinoma was made by Auerbach et al.[67] Carcinoma in situ occurred in 89% of the 54 cases. The carcinoma in situ slides were divided into two groups: (1) those with definite carcinoma in situ in which all of the accepted criteria for the diagnosis were present with an incidence of 9.2%, and (2) borderline carcinoma in situ cases where one or two of the criteria were absent with an incidence of 7.9%. The carcinoma in situ changes

were widely distributed throughout all segments of the tracheobronchial tree although somewhat irregularly present, and there was a comparable incidence of these lesions in the opposite lung as well as in the lung involved by the obvious bronchial carcinoma. Early invasive carcinoma was observed in 5 of the 54 cases and suggested that carcinoma in situ is a stage prior to invasion.

Among 144 unselected autopsied patients, Valentine [68] found the average incidence of squamous metaplasia of the bronchus to be 32.8% in comparison with an average incidence of 61.9% in patients dying of chronic pulmonary disease and 56.5% in patients with bronchogenic carcinoma. In three surgically removed lungs with bronchogenic carcinoma both squamous metaplasia and intraepithelial carcinoma were found adjacent to the invasive tumor. One case of carcinoma in situ was discovered in the group of 144 unselected noncancerous patients.

Steele [69] reviewed 228 proved cases of bronchogenic carcinoma in which bronchoscopic examination was performed. There were 103 cases of the squamous-cell type, 77 cases of undifferentiated carcinoma, and 48 patients with adenocarcinoma. In 97% of the patients bronchogenic carcinoma was suspected on the roentgenogram. The diagnosis was made by bronchoscopic biopsy in 40.4% of the patients, while 36.8% demonstrated pathologic changes in the tracheobronchial tree suggestive of bronchogenic carcinoma. Cytologic studies of bronchial secretions were performed in 61 patients with the presence of malignant cells noted in 47.5%. A higher percentage of positive cytologic examinations for malignant cells was made possible by abrasion of the bronchial wall with bronchoscopic forceps or aspirating tubes followed by forceful washing of such areas with saline solution. With use of this technique the percentage of positive cytologic diagnoses increased to 55.5% while biopsy was positive in only 25.9%.

In a series of 42 histologically proved lung cancers Umiker and his associates [70] reported the diagnosis was made by bronchoscopic biopsy in 40%, by anterior scalene lymph node biopsy in 24%, and by cytologic examination of sputum or bronchial aspirate in 69% (positive) or 88% (positive or suspicious). Bronchoscopic biopsy was diagnostic most often in the squamous cell type and in carcinomas in main-stem or lower lobe bronchi. Positive cytologic findings were the only signs of malignancy in 26% of the patients, and was successful in detecting the majority of all type of bronchogenic cancers, including upper lobe and peripheral lesions. Positive cytologic results, together with the clinical findings even in the absence of other microscopic evidence, proved reliable in detecting carcinoma. The resectability of lesions diagnosed by bronchoscopic biopsy or cytologic study was considerably less than those with negative results. One false positive cytologic diagnosis was obtained.

Dysphagia

Foxen [71] described a case in which esophagoscopy for dysphagia resulting from cricopharyngeal spasm led to the finding of an islet of gastric mucosa at the beginning of the esophagus. This isolated area of ectopic gastric mucosa in the upper esophagus was thought to cause dysphagia by reflex cricopharyngeal spasm.

Esophagoscopy in Upper Gastrointestinal Bleeding

In a series of 109 patients with massive upper gastrointestinal hemorrhage Smythe et al.[72] performed emergency esophagoscopy in 24 cases. In 9 no lesion was found and in 12 varices were seen, with active bleeding apparent in 3. Probable varices were found in two other cases and a carcinoma in one. Difficulty was encountered in positively identifying a varix in a blood-filled esophagus, so as an alternative esophageal balloon tamponage as a diagnostic measure was employed. There were no complications of

diagnostic esophagoscopy, and in no case was bleeding reactivated.

Pneumonitis in Esophageal Diseases

The high incidence of the aspiration type of pneumonitis in esophageal disease was pointed out by Vivas and Palmer.[73] In a group of 100 cases chronic pulmonary disease was found in 22. A further breakdown indicated that these changes occurred in 2 of 42 patients with hiatus hernia without secondary esophageal change, in 4 of 13 with diverticulum of the esophagus, in 2 of 12 with subacute erosive esophagitis without hiatus hernia, in 6 of 8 with achalasia, in 3 of 7 with subacute erosive esophagitis with hiatus hernia, in 2 of 3 with carcinoma of the esophagus, and in 1 of 3 with a pharyngoesophageal diverticulum. Two patients with esophageal stricture due to swallowing lye had chronic pulmonary disease, while none of the 10 patients with other forms of esophageal strictures had pulmonary involvement, and these 10 included 6 with stricture secondary to esophagitis, 3 with congenital stricture, and 1 with radiation stricture.

Foreign Bodies in the Esophagus

Jackson[74] reported his experiences with foreign bodies in the esophagus and found that about half occurred in males and half in children. The largest group of foreign bodies were bones, followed by coins, food, pins, vegetable foreign bodies, dental objects, buttons, and toys. Among the etiologic factors the wearing of artificial dentures was a predisposing cause because it interfered with the detection of a foreign object such as a bone when in the mouth. Carelessness in the preparation of food and hasty or careless eating was another factor. The placing of inedible objects in the mouth was condemned. Stenosing lesions of the esophagus predisposed to the lodgment of an esophageal foreign body, and because of this possibility all cases had a roentgen film made after the foreign body was removed to rule out this possibility. The treatment consisted of removal, and the technique employed was described. Almost all foreign bodies lodged in the esophagus were removed esophagoscopically and only the rare case required esophagotomy for removal of the object.

Zoller and Bowie[75] called attention to the fact that normal calcification of isolated portions of the laryngeal cartilages may be superimposed on the food passages and on roentgen examination give the impression of a foreign body. The patient's history and a thorough examination, including lateral roentgenograms of the neck, should be performed prior to esophagoscopy, and if the physician is familiar with the normal calcification shadows needless esophagoscopy may be prevented. If the symptoms persist esophagoscopy has to be performed regardless of the x-ray findings.

Perrone and Cipcic[76] reported the removal of a foreign body in a 7-month-old premature infant by esophagoscopy. The chief findings were dyspnea and cyanosis becoming increasingly more marked from its onset on the fifth postpartum day. Roentgen examination revealed a piece of rubber catheter lodged in the midesophagus which was responsible for the respiratory difficulty because of pressure on the tracheobronchial tree. At esophagoscopy the foreign body, which was swallowed after a piece of catheter fitted onto the end of a medicine dropper used for feeding became disloged and swallowed, was removed.

Nichamin and Gonne[77] reported a case of a foreign body (paper clip) in the upper esophagus of an infant who had respiratory symptoms present since birth and no related foreign body history.

Peeler and Riley,[78] observed a year-old-child in whom a swallowed safety pin perforated through the esophagus into the heart, producing cardiac tamponade. There was no foreign body history, but the illness dated for about eight weeks. After hospital admission the foreign body (open safety-pin) was found on roentgen examination lodged in the lower esophagus with the point in close proximity and possibly within

the cardiac silhouette, with other changes suggesting mediastinitis. The pin was removed at esophagoscopy by endogastric version, but the case terminated fatally 11 days after pericardiostomy for pericardial effusion and infection.

The external approach for the removal of coins from the esophagus was presented by Sanderson.[79] It is difficult to rationalize this approach when foreign bodies of this type present no particular problem of removal by esophagoscopy.

Equen et al.[80] reported on the magnetic removal of 14 open safety pins and 14 other objects from the esophagus, while in 189 cases various objects were removed from the stomach and 19 foreign bodies were taken from the duodenum. There were no deaths in the series, but in two other patients perforation of the duodenum required surgical exploration, both of whom recovered.

Diverticulum of the Esophagus

In discussing 75 cases of esophageal diverticula Mustard[81] found 53 of the pharyngeal type, 14 epibronchial, and 8 epiphrenic. The cases of pharyngeal diverticula were operated upon with 3 deaths and 3 recurrences. The symptoms of epibronchial diverticula were dysphagia, chest pain, and epigastric distress, and only one case was operated upon for excision of the sac. The symptoms of epiphrenic diverticula were regurgitation, dysphagia, chest pain, and hematemesis; six cases were operated upon and the sac excised, with one death, and one case was treated by dilatation.

McBurney[82] reported a series of 57 patients with esophageal diverticula, 32 being of the pulsion type of pharyngoesophageal diverticula, 17 the midthoracic traction type, and 8 the epiphrenic type. The diagnosis was made by roentgen examination, and esophagoscopy was condemned because of the danger of perforation which the author felt far outweighed the value of the information obtained.

Lodge[83] reported on the excision and pari-passu repair of pharyngeal diverticula in six cases. An external approach was utilized, and the pharyngeal opening was closed in a series of equal steps passing the suture from the mucosa to the muscularis of one side and from the muscularis back to the mucosa of the other so that the knots lay in the lumen. A second row of sutures passing through the muscular coat only was used to invaginate the first row. The cases so treated were advanced, and the healing was prompt.

Webster[84] reported a case of spontaneous perforation of a lower esophageal diverticulum following ingestion of a heavy meal. There was also an associated hiatal hernia but the perforation occurred through the diverticular sac.

Neuromuscular Imbalance of Esophagus

Atkinson et al.[85] studied the physiology of swallowing by means of intrapharyngeal pressure measurements with electromanometers. The resting intraluminal pressure was elevated at the pharyngoesophageal junction in each of the 30 normal subjects studied and the mean pressure level was 35 mm. of mercury. This zone of elevated resting pressure corresponded roentgenographically with the position of the cricopharyngeal sphincter. During swallowing in the high pressure zone at the cricoid level an abrupt fall to or to just above intraesophageal pressure occurred on swallowing followed by an equally abrupt return to the high resting level after the passage of the bolus. These findings supported the view that the cricopharyngeus was a true sphincter and swallowing did not involve suction.

Cross and his associates[86] reported an over-all study of 60 patients with esophageal problems by cinefluorography and intraesophageal pressure recordings. In conditions of neuromuscular imbalance with the same clinical picture of esophageal obstruction and dysphagia two distinct entities which were either primary or related to some precipitating factor were found. Neuromuscular failure was characterized by

a hypoactive esophagus and was seen in older people, whereas in segmentation the esophagus was hyperactive and was found at all ages.

Neuromuscular disorders involving the swallowing mechanism were investigated by Kramer et al.[87] Four patients with postpoliomyelitic dysphasia, two with myasthenia gravis, and one with dystrophia myotonica were studied by recording the intraluminal pressures. After poliomyelitis failure to close off the nasal passages with paralysis of the pharyngeal constrictor muscles appeared to be the principal abnormality, and the cricopharyngeus functioned normally in each of the four patients. In the other patients with myasthenia gravis and dystrophia myotonica generalized muscular weakness appeared to be an important factor, but the cricopharyngeus muscle was also involved in this disability.

The use of cinefluorography and intraluminal pressure tracings for evaluating neuromuscular changes in the esophagus in association with hiatus hernias was used by Cross and his associates.[88] These studies proved valuable as investigative diagnostic and teaching aides in neuromuscular diseases of the esophagus, and the basic pattern obtained in patients with hiatal hernia was hypoactivity of the esophagus, elevated base pressures, and hyperactivity of the inferior esophageal sphincter.

Ardran and his associates [89] demonstrated radiographically that the swallowing difficulty in eight patients following bulbar poliomyelitis was due to failure of the muscles controlling the lower end of the lateral food channels and the opening of the esophagus to relax. In all cases the middle pharyngeal constrictor muscles were considerably weakened, and the patients used all possible means to reduce the size of the pharyngeal cavity to its smallest dimensions.

A surgical procedure for the restoration of swallowing was performed by Kaplan [90] on eight patients who had lost this function as a result of bulbar poliomyelitis. The operation was done under local anesthesia in adults and consisted essentially of sectioning the cricopharyngeus muscle on one side only. This was usually done on the left side, and the section was carried through the muscle down to the mucosa. There was one failure, but a secondary complication of tongue block delayed recovery in some cases.

Esophageal Web

Bugden [91] described a new entity causing lower esophageal obstruction, namely, lower esophageal web. The roentgen findings were similar to those of a short stricture or contractile esophageal ring. In the roentgen studies thick barium was advised, for thin material did not delineate the lesion and passed through the area without hesitation. Endoscopic examination may be misleading, for the web may not be visualized and the examiner may be uncertain whether the esophagogastric junction is anatomically difficult to pass or whether there is a true organic obstruction. Of the five cases reported the first two were misdiagnosed endoscopically and the others were characterized by a rather marked sudden rigid shelf, at times with an eccentric lumen approximately at the diaphragm level. Little data exist as to the true nature of the lower esophageal web, but the author treated all cases by surgery.

Four cases of esophageal webs diagnosed by roentgenography and confirmed by esophagoscopy in two cases were reported by Waldman and Turnbull [92] None of the patients had the Plummer-Vinson syndrome, and the webs were all located in the region of the cricopharyngeus muscle. Two cases responded satisfactorily to dilatation while the others required no mechanical treatment.

Perforation of Esophagus

Fifty-four cases of traumatic perforation of the esophagus were reported by Mathewson et al.[93] Of these 18 resulted from trauma induced by the esophagoscope; 15 were caused by the ingestion of foreign bodies; 6 were secondary to attempted

dilatation; 4 were secondary to biopsy; 1 was caused by gastroscopy, and 2 were due to external trauma. The site most commonly injured at endoscopy was at the level of the cricoid cartilage in the region of the cricopharyngeus. The second most common point was just above the constriction where the esophagus passes through the diaphragm, and the third site was the narrowing at the level of the aortic arch and left main bronchus. Even though the endoscopist frequently recognizes a perforation as it occurs, unrecognized trauma should also be kept in mind after endoscopic examination. Pain was the most prominent symptom encountered. Fever and difficulty swallowing were common early symptoms but these also depended upon the extent and location of the perforation and the amount of inflammation present. Respiratory distress, hoarseness, and cyanosis were encountered to a slighter degree. In perforation of the cervical esophagus local tenderness and crepitation of the soft tissues were early signs, whereas dysphasia, hoarseness, and pain developed later. Perforations with direct entry into the pleural cavity were followed usually by accummulation of fluid and air in the pleural space with an occasional tension pneumothorax. Ruptures of the lower esophagus were associated with severe substernal and epigastric pain, dyspnea, and cyanosis, and these patients were all severely ill from the onset and were usually in a state of shock very soon after the injury. The usual radiologic findings consisted of mediastinitis, pleuritis, hydropneumothorax, and mediastinal emphysema. Radio-opaque material was given by mouth in several cases, and the diagnosis was confirmed. Conservative management was undertaken in 25 patients, with 5 deaths, while in the entire group of 54 cases there were 11 deaths. In analyzying the deaths it became apparent that patients with laceration of the esophagus involving the pleura did poorly on conservative management, but those with lacerations involving the mediastinum alone recovered without surgical intervention. In one of the small children perforation during dilatation of a stricture of the thoracic esophagus was recognized immediately, and open thoracotomy with repair of the esophageal laceration resulted in an uncomplicated convalescence. In another infant traumatic perforation was seen through the esophagoscope before completion of the endoscopic examination, and immediate thoracotomy and repair of the laceration led to an uncomplicated recovery. In contrast, there were two other patients under one year of age each of whom suffered perforation of the thoracic esophagus following dilatation that was not suspected until high fever, restlessness, and signs of pneumothorax led to the diagnosis, and closed drainage of the chest did not prevent death.

Chamberlain and Byerly[94] found that instrumental trauma was the commonest cause of perforation of the esophagus. Patients with small cervical esophageal rupture in the early stages were treated by appropriate antibiotic therapy when they were seen immediately or when there was no evidence of gross contamination. Large cervical esophageal ruptures in the early stages were treated by primary suture, while large or small cervical esophageal ruptures in the late stages after there had been cervical mediastinal contamination with abscess formation were treated by cervical incision and drainage. Large and small ruptures of the thoracic esophagus in the early stages were best treated by early open operation which was also applicable for drainage purposes to those seen in the late stages. If the patient was in poor condition, it was necessary to stage the procedure for drainage. Careful attention was also given to oral hygiene, and antibiotics were administered by lozenge in order to reduce the bacterial flora in the mouth along with systemic administration of the drug which was carried out in all cases.

In two cases in which esophageal perforation occurred during an attempt to instrumentally administer therapy the diagnosis was made by Blalock[95] on a clinical basis. No radiologic proof of perforation was ob-

tained for the diagnosis was not in doubt. Primary resection produced satisfactory results in these two patients in whom resection for low esophageal lesions would ultimately have been necessary anyway. In the cases reported the perforations were in the lower esophagus, and early operation was advocated in those patients with extensive esophageal laceration and gross contamination of the mediastinum and pleura.

Brewster[96] described an unusual case of traumatic perforation of the esophagus due to self catheterization by the passage of a heavy electric wire into the esophagus. The patient swallowed a large portion of meat which stuck in his throat, and when attempts to dislodge the meat by coughing and drinking water were unsuccessful he passed a length of heavy electric wire down his throat in the manner identical with the sword-swallowing act. The bolus of meat was pushed down the esophagus into the stomach, but on completion of the self-instrumentation the patient suddenly experienced severe substernal pain with developing dyspnea. Conservative therapy with adequate antibiotic therapy and nothing by mouth were employed with successful recovery. It was later disclosed that the patient had a habit of bolting his food with frequent impaction of meat in the esophagus and had performed the procedure approximately 200 times over a 20-year period successfully with no ill effects.

Anderson[97] discussed spontaneous rupture of the esophagus, and of the 50 cases esophageal ulceration was considered the etiologic agent in 11. The other cases were produced either by increased intraluminal pressure as a result of vomiting or spontaneously without an obvious cause. The site of the rupture was most commonly found at the lower end on the left posterior lateral wall, and immediate thoracotomy was advocated with closure of the rupture as the primary treatment.

Naclerio[98] described a V sign on the roentgenogram in cases of spontaneous rupture of the esophagus. The sign consisted of a localized mediastinal emphysema in the lower mediastinum, which together with a characteristic history was diagnostic.

Stenosis of the Esophagus

Benedict and Nardi[99] considered the prognosis in benign stenosis of the esophagus very good as regards palliation by bouginage. In this condition direct inspection by esophagoscopy revealed a smooth conical narrowing with varying degrees of inflammation manifested by reddening, edema, and often erosions and superficial ulcerations. Esophagitis was found to be extensive, in some cases extending 10 to 15 cm. above the area of narrowing. At times foreign bodies, particularly vegetables, lodged at the site of stenosis. Biopsy was performed at the area of narrowing to exclude carcinoma. The treatment of benign stenosis by bouginage was advocated except in certain intractable cases. Bouginage was performed with metal olive tips over a previously swallowed string, the limit of three bougies at one treatment being adhered to. When resistance was encountered a smaller size bougie was used, and if bleeding occurred excessively the dilatation was temporarily discontinued. The dilatation was carried up to No. 35 F. and patients were able to eat satisfactorily. Surgery was performed in a limited number of cases and generally gave good results when meticulous care was used in establishing the anastomosis, but some recurrences of the inflammatory stenosis at the esophagogastric junction were encountered.

Esophagitis

Hale and Drapanas[100] discussed the etiology and clinical picture of reflux esophagitis on the basis of their experience with 102 cases. Esophagoscopy was considered an integral part of the diagnosis, revealing the degree of esophagitis, ulceration or stenosis, accurate establishment of the level of the cardioesophageal junction, determination of the amount of fixation of the lower esophagus to the mediastinum, and elimination of malignancy by biopsy if

necessary. On esophagoscopy the characteristic changes of hyperemic lower esophageal mucosa with bleeding tendencies and prominent folds were observed frequently associated with small ulcerations. A relaxed cardia with lack of deviation was found in those cases not demonstrated by roentgenography. The following factors were considered active in the mechanism of reflux esophagitis: (1) incompetence of the cardioesophageal sphincter, (2) gastric hypersecretion, (3) protracted vomiting, (4) ectopic gastric mucosa, and (5) individual susceptibility. In the series several patients had esophagitis on esophagoscopic examination with bizarre or minimal symptoms. Hemorrhage was prominent in 8 patients and obstructive symptoms were noted in 21. Sixty of the cases were treated surgically and repair of the hernia was generally employed first. With the persistence of symptoms and esophagitis subtotal gastrectomy was done, and in a few instances of fibrous stenosis esophageal resection was resorted to.

Twenty-three patients with primary esophageal ulcer were reported by Palmer.[101] Associated diseases of the gastrointestinal tract were frequent, and the chief symptoms were weight loss, painful and difficult swallowing, and bleeding. Diagnosis by esophagoscopy was more accurate than by roentgenography. The ulcers had uneven bases and in two cases polypoid changes were found along the crater edges. Besides oral medication esophagoscopic dilatations were used in the treatment; the average healing time was four weeks. Esophageal dilatations were carried out about twice a week for the first month using mercury bougies up to No. 48 F. The usefulness of dilatations could not be explained, and its value was thought to lie in symptomatic relief and encouragement of healing possibly by breaking up segmental spasm, but this explanation was not convincing.

A case of "gastric lined" esophagus with esophagitis was reported by Som and his associates.[102] The diagnosis was suggested on roentgenography and confirmed by esophagoscopy and serial biopsies taken from graduated levels. Esophagitis in specimens taken 30 cm., 34 cm., and 40 cm. respectively from the alveolar margin was found at the superior and inferior margins of the gastric-lined region.

Mustard[103] emphasized that the diagnosis of esophagitis can only be made with certainty by esophagoscopic examination.

In discussing corrosive esophagitis Daly and Cardona[104] divided the clinical picture into three groups: mild, severe and severe complications. Esophagoscopy was used to accurately determine the extent and severity of the burn and also as a means of following the progress of healing. The esophagoscope was passed to the first burned area but not beyond it, so that no complications developed. After neutralization of the caustic, cortisone and antibiotics were used to reduce the local reaction in the esophagus and indirectly reduce the tendency to stricture formation. Early dilatation with weighted bougies maintained the lumen and prevented adhesions between the ulcerated areas in the esophageal wall. Dilatation was carried out either perorally or retrogradely through a gastrostomy. Surgical treatment was employed in strictures with complicated adhesions, irregularities or periesophageal reactions, fistulas or complete stenosis.

Gastroesophageal Incompetence

Tatelman and Burton[105] reported a case of gastroesophageal incompetence in a newborn which proved to be the first stage in the development of a hiatal hernia about three weeks later.

Stensrud[106] reported a case of incompetence of the cardia with reflux of gastric juice in the esophagus treated by a minor plastic surgical procedure. On esophagoscopy an inflamed thickened mucous membrane in the lower part of the esophagus with severe regurgitation but no ulceration or actual hiatus hernia was observed. Postoperative esophagoscopy revealed slight esophagitis but no regurgitation.

Davis[107] described the anatomy of the sphincters and their action in the closure of

the cardiac orifice of the esophagus. Observations were made roentgenologically by barium swallow and esophagoscopically in animals and fresh cadaver specimens. The inferior esophageal sphincter was found 1-2 cm. above the diaphragmatic hiatus, and the mucosa was thrown into folds as part of the esophageal sphincter.

Hiatal Hernia and Esophagitis

Lindskog and Kline [108] reported 12 cases of peptic esophagitis complicating the sliding type of hiatus hernia proved by esophagoscopy. The earliest changes of esophagitis were found only on endoscopic examination, and the esophagogram appeared normal until the process was far advanced and the distal esophagus had become thickened and shortened by fibrosis and the mucosa grayish-white in color. At this stage the changes were indistinguishable on the roentgenogram from neoplasm, and esophagoscopy was again needed to differentiate these two conditions. The earliest esophagoscopic changes of esophagitis consisted of epithelial necrosis and edema with or without discreet shallow ulcerations.

Reid and Robinson [109] presented a case of hiatus hernia where the lower esophagus was lined with gastric mucosa and a stricture from esophagitis was found above this level. The stricture was about 1-2 cm. long at the level of the aortic arch, and gastric mucosa was situated immediately below this region.

Esophageal Hiatal Hernia

By means of electromanometric measurements Marchand [110] studied the forces producing gastroesophageal regurgitation and hiatus herniation. This experimental work was done on six persons, and the hiatus was found to move on an average of 2 cm. downwards during each deep inspiration. During quiet respiration in the supine position the average gastric pressure fluctuated from plus 2 cm. of water on expiration to plus 10 cm. of water on inspiration. On coughing considerable pressure was momentarily generated as it did when the abdominal muscles were tensed. It was thought that the intragastric pressure was the force which produced gastroesophageal regurgitation. This was influenced by posture, respiration, coughing, and straining. In hiatus hernia degenerative changes due to age alone cannot always be implicated, and structural weaknesses or softening may predispose to the development of a hernia in the face of increased abdominal pressure. It was believed that the same stress which produced regurgitation and gastroesophageal reflex also in time caused disruption of the anterior hiatal muscles, thus initiating the development of a hiatus hernia.

The sphincteric mechanism at the esophagogastric junction was studied by manometry in normal subjects and in patients with hiatus hernia by Atkinson and his associates.[111] The findings indicated that the sphincter is intrinsic and is not caused by external compression from the diaphragm. The pressure exerted by the sphincter at the esophagogastric junction constituted a barrier to gastroesophageal reflux, and the tone of the sphincter prevented reflux.

Barrett [112] discussed the lower esophagus lined by columnar epithelium and considered it a result of failure of the embryonic lining to mature. Distinction between this condition and sliding hiatal hernia was made. On esophagoscopy the diagnosis was suspected if the change of mucous membrane was found at a high level. Since the epithelial transition was sharp, no difficulty, was encountered in marking the demarcation unless local inflammation was present. In most cases the change occurred higher than would be expected in a sliding hiatal hernia and did not coincide with the level of the cardia radiologically. On the average the change-over was noted about 20 cm. from the incisor teeth and, when doubtful, pinch biopsies were diagnostic. The finding of columnar epithelium above a pathologic lesion was used to differentiate a benign from a malignant stricture at the esophago-

gastric junction provided adequate tissue was studied histologically and carcinoma was not found.

Sixty-three cases of hiatus hernia in children were presented by Engberg and his associates.[113] The high incidence of hiatus hernia in children was attributed to more frequent use of roentgen examinations in unexplained cases of regurgitation and vomiting. Conservative measures were satisfactory in many cases, especially in those without fibrous stenosis. When esophagitis existed it could not be treated successfully by simple surgical measures, and esophageal resection was performed even though there was high mortality and postoperative sequelae. Resection was postponed as long as possible and only utilized when other measures failed. Operative treatment was recommended in all cases of paraesophageal hiatus hernia because of the danger of compression to adjacent thoracic organs, incarceration, or ulceration.

Six patients with short esophagus and thoracic stomach in infancy were followed for a period of over 20 years by Kelly.[114] All of the patients led normal lives with slight symptoms of regurgitation. Primary treatment consisted of a short course of bouginage with no subsequent therapy. In two cases the short esophagus and thoracic stomach were demonstrated in early childhood both by roentgen and esophagoscopic examinations, but 20 years later no evidence of thoracic stomach was found radiologically even though symptoms of incompetence of the cardiac mechanism and esophagitis were still present.

The indications for esophagoscopy in esophageal hiatus hernia were listed by Buchanan et al.[115] as severe anemia, pain, dysphagia, patients with symptoms and evidence of hemorrhage and in those with roentgen signs of obstruction or stricture when surgical treatment was being considered. Esophagoscopy permitted accurate evaluation of coexisting esophagitis, ulceration, and obstructions, and carcinoma or scleroderma might be detected. Difficulty was found in distinguishing inflammatory narrowing of the lumen from submucosal fibrosis.

Fisher and Johnson[116] realized the usefulness of esophagoscopy in hiatal hernia and recommended that the examination be done routinely regardless of the roentgen findings. The most important factor in the etiology of peptic esophagitis was found to be incompetence of the valve-like action at the esophagogastric junction. On esophagoscopy abnormality of the mucosa, regurgitation through the hiatus, redundancy of the mucosa, patency of the cardiac orifice, and a loss of the acute angle of entry of the esophagus into the stomach were demonstrated.

MacDougall and Abbott[117] stressed the important role of esophagoscopy in the diagnosis of hiatus hernia. In the 108 cases listed the diagnosis was established on esophagoscopy in 6 cases in which the roentgenograms were reported as normal.

Phear[118] surveyed 87 cases of hiatus hernia and 20 of gastroesophageal reflux. Esophagitis was found radiologically in only four cases, while esophagoscopy in one of these revealed a normal esophageal mucosa even with reflux gastric regurgitation. In five other patients examined esophagoscopically the mucosa was normal in three, hyperemic in one, and ulcerated in one. The incidence of esophagitis in this series was low because esophagoscopy was not performed on all patients and mild degrees of esophagitis cannot be demonstrated radiologically. Medical treatment produced good results in 70% of these cases and considerable relief in an additional 14%.

Maurer and Keirle[119] reported their results in the surgical repair of 57 cases of esophageal hiatus hernia and emphasized the need for preoperative esophagoscopic examination to exclude complicating carcinoma, ulcer or stricture. Esophagoscopy was also indicated if symptoms of this lesion persisted despite normal barium swallowing function examination. Exact localization of the esophagogastric junction together with information concerning the

condition of the esophageal mucosa and the presence or absence of esophagitis proved valuable.

Two cases of carcinoma of the stomach associated with hiatus hernia were reported by Friedman.[120]

Hiatal Hernia and Achalasia

A case of coexistent achalasia and hiatal hernia with lower esophageal obstruction was reported by Euphrat and Delmonico.[121] The esophagoscopic findings revealed marked dilatation and tortuosity of the esophagus with obstruction proximal to the cardia from moderate inflammation and edema, preventing passage of the esophagoscope into the stomach.

Achalasia

Olsen et al.[122] demonstrated by motility studies of the esophagus of patients with achalasia that therapeutic procedures available at the present time do not restore the motility of the esophagus. The suprahiatal portion of the inferior esophageal sphincter was usually abolished by either hydrostatic dilatation or esophagomyotomy and this reduced the resistance of the sphincter sufficiently to allow a more normal passage of esophageal contents into the stomach. Retention of the inferior hiatal portion of the sphincter apparently prevented reflux in these patients. This study revealed that both hydrostatic dilatation and cardiomyotomy were effective but not ideal methods of treating cardiospasm because neither procedure restored peristalsis in the esophagus. Both were successful in reducing the resistance which must be overcome at the lower end of the esophagus without completely destroying the sphincteric mechanism.

Pressures during resting and deglutition were measured at the gastroesophageal and pharyngoesophageal junctions in 13 cases of achalasia by Creamer and his associates.[123] The obstruction at the gastroesophageal junction was produced by failure of the sphincter to relax during swallowing and a loss of the normal propulsive peristaltic wave throughout the body of the esophagus. It was also found that the sphincter contracted normally at the end of swallowing but earlier than in health so that the pause between the beginning of swallowing and the sphincteric contraction was shortened. Their findings demonstrated that the resting tone of the gastroesophageal sphincter was not increased in achalasia and was of normal proportion, but the sphincter failed to relax during deglutition. The action of the cricopharyngeal sphincter in achalasia was normal.

Olsen and Creamer[124] considered achalasia and diffuse spasm of the esophagus to be separate entities and usually differentiated by clinical, radiological, and endoscopic means. Esophageal motility studies most accurately differentiated these two conditions. Similarities between cases of early cardiospasm and diffuse spasm required esophageal pressure studies and the mecholyl test for differentiation. These studies indicated that in diffuse spasm the lower esophageal sphincter at rest has a wider band of high pressure than it does in achalasia. On deglutition diffuse spasm as indicated in the upper esophageal sphincter showed simultaneous repetitious contractions with high pressures, while in achalasia there was complete failure or feeble contractions. On deglutition the lower sphincter in diffuse spasm revealed relaxation and then contraction which occurred vigorously, while in achalasia there was no relaxation observed.

Muscle strips from the lower end of the esophagus of a group of patients with achalasia of the cardia were studied by Traunce et al.[125] and compared with muscle from similar regions in patients without achalasia. In both groups the muscle behaved as smooth muscle; a mechanism involving cholinesterase was present, and active ganglion cells were found in the majority. Although the histologic picture was variable, approximately normal numbers of ganglion cells were found in two out of six patients with achalasia.

Olsen and his associates [126] reported a series of 601 patients with cardiospasm, 555 of whom were treated by means of hydrostatic dilatation. In this latter group 10 had esophageal rupture, constituting a complication rate of 2%. Long-lasting and permanent relief was obtained in 60% of the patients with a single course of hydrostatic dilatation and ultimately was achieved after two or more courses in 80% of the cases. Dilatation was useful in early lesions, especially when pain was prominent. When cases did not respond to conservative management and hydrostatic dilatation, surgical intervention was employed in 37 patients by means of a modified Heller procedure. Of the 27 patients available for follow-up 16 had excellent results, while 3 had fair results with improvement but had either recurrence or persistence of dysphasia, and one patient developed severe postoperative esophagitis.

Three cases of achalasia in which the dilated esophagus suggested the presence of a mediastinal tumor and the diagnostic difficulties encountered were described by Moser and McQuiston.[127]

Esophageal Varices

Palmer [128] performed serial esophagoscopic examinations with portal pressure determinations in some cases in a group of 133 patients with esophageal varices and cirrhosis of the liver. It was found that the varices changed in diameter and extent from time to time with only the slightest tendency to improve as the general clinical course improved and to worsen as the clinical course worsened. Increasing stability was noted as the varices became larger and more extensive. No correlation between the duration of the cirrhosis and the severity or stability of the varix could be determined. In 25% of the patients one examination did not reveal varices but subsequent examinations revealed their presence. The pressure in the portal venous system could not be correlated with the diameter or extent of the varices nor could it be estimated from the esophagoscopic appearances. The pressure varied considerably from time to time with no relation to the clinical picture, but in the individual patient it was found that varices did not become smaller when the pressure fell or larger when it rose.

Palmer [129] followed 63 patients with cirrhosis of the liver by periodic esophagoscopic examinations following surgical portal decompression for esophageal varices. Another group of 45 patients with proved cirrhosis in whom the shunt operation was recommended but who refused operation was used as a control series. After portacaval shunt approximately 40% no longer had varices one month later, and with the passage of time the varices became worse in a very small number. In those patients treated by splenorenal shunt the initial postoperative examination demonstrated considerable improvement, but thereafter 59% had progressive enlargement of the varices. Among the control patients 73% of the varices were either unchanged or worse. In six instances of the latter group the varices disappeared spontaneously and returned later in three. The conclusions indicated that portacaval shunt was far more effective in reducing portal pressure and eliminating varices than was splenorenal shunt.

Benign Tumors of Esophagus

Boyd and Hill [130] reported 17 cases of benign tumors and cysts of the esophagus in which only 7 had complaints specifically referred to the esophagus. The diagnosis was established roentgenologically, and biopsy of the esophagus was considered unnecessary and undesirable. External surgical removal was advocated in all cases except polyps. In polyps the treatment of choice was esophagoscopy and snare removal, which was carried out in the one case reported.

A case of lipoma of the esophagus, believed to be the 10th reported, was presented by Ochsner and Ochsner.[131] Both roentgenologically and esophagoscopically the lesion was thought to be carcinoma because of its indurated constricting appearance just

above the esophagogastric junction. Tissue removed for histologic study was reported as subacute inflammation with no tumor present. The submucosal lesion was removed by enucleation via external thoracotomy. It was noted that occasionally this type of lesion might be removed by endoscopic snare if pedunculated.

Malignant Tumors of the Esophagus

Carter [132] described a technique of endoscopic insertion of polyethylene tubes for palliation of obstructing esophageal carcinoma. Plastic tubes were inserted in 19 of 24 patients in whom it was considered, and successful palliation was achieved in 13. In five of the failures complications developed which together with the advanced state of the tumor resulted in death five days to one month later. The patients in whom intubation was successfully carried out swallowed liquids and semisolids satisfactorily, living from 1 to 12 months, and one patient remained well for 36 months after intubation and radiation therapy. Esophageal dilatation was also used to maintain a lumen adequate enough for solid foods to pass in patients who had completed a course of radiation therapy and in whom narrowing due to fibrosis or recurrent tumor developed. In the successfully intubated patients there was noted an abrupt change from terminal illness with aspirational pneumonia to normal temperature, adequate hydration, and swallowing comfort. The tubes were made of polyethylene with flanging on one end obtained after heating slightly and measured approximately 0.6 cm. in external diameter with an internal diameter of 0.5 cm. The upper end was marked for subsequent roentgen examination by wrapping several turns of stainless steel wire distal to the flange. Under general anesthesia a 9×45 cm. esophagoscope was passed, except in high lesions when a shorter instrument was used. After location of the tumor secretions and food were removed following which a small flexible Jackson dilator was carefully insinuated through the lumen. The length of the strictured area was estimated and the polyethylene tube cut to the desired length. With the flanged end uppermost the tube was passed through the esophagoscope over a previously inserted No. 10 or No. 12 Jackson dilator and gently pushed into position with a suction tip. With the dilator acting as a guide the tube was advanced by utilizing the tip of the esophagoscope until the flange rested above the protruding tumor mass. Occasionally foreign-body forceps were needed to place the tube accurately. The following day roentgen films were made both with and without iodized oil and the metal wiring around the upper end of the tube was used for localization. On occasion the tube slipped and passed through the intestinal tract, but it rarely became plugged with solid food requiring esophagoscopy for removal of this material.

A case of primary malignant melanoma of the esophagus was reported by Keeley et al.[133] Esophagoscopy revealed an irregular, friable mass 30 cm. from the incisor teeth, which bled readily when tissue was removed. The pathologic diagnosis was sarcoma of undetermined origin on the biopsy specimen.

1712 Locust St.

REFERENCES

1. Hilding, A. C.: Ciliary Streaming in the Bronchial Tree and the Time Element in Carcinogenesis, New England J. Med. 256:634 (April 4) 1957.

2. Dekker, E., and Groen, J.: Asthmatic Wheezing, Lancet 1:1064 (May 25) 1957.

3. Deloyers, L.; Cordier, R., and Duprez, A.: A New Approach to the Physiology of So-Called "Cardiospasm," Ann. Surg. 146:167 (Aug.) 1957.

4. Muren, A.: Experimental Oesophageal Fistula with Sphincter Function, Acta. chir. scandinav. 112:92 (Feb.) 1957.

5. Jackson, C. L.: New Instrumentarium for Peroral Endoscopy and Endoscopic Photography, Laryngoscope 67:777 (Aug.) 1957.

6. Holinger, P. H., and Johnston, K. C.: Bronchoscopy and Endoscopic Photography, S. Clin. North America, p. 1311, Oct. 1957.

7. Soulas, A.; Dubois De Montreynaud, J. M.; Edwards, R. J., and Gladu, A. J.: Bronchoscopy and Television, Dis. Chest 31:580 (May) 1957.

8. Cracovaner, A. J.: Changing Concepts in Bronchology, A. M. A. Arch. Otolaryng. 65:547 (June) 1957.

9. Som, M. L., and Arnold, L. M.: Esophagoscopy in the Diagnosis and Treatment of Esophageal Diseases, Am. J. Surg. 93:183 (Feb.) 1957.

10. Wyant, G. M.; Dobkin, A. B., and Kilduff, C. J.: Problems of Anesthesia for Bronchoscopy, Canad. M. A. J. 76:1011 (June 15) 1957.

11. Hughes, F. A.; Mobley, J. D.; Bills, R. J.; Burwell, J. R., and Pate, J. W.: An Evaluation of 2 New Topical Anesthetic Agents: Dyclone and Cyclaine, J. Thoracic Surg. 33:685 (May) 1957.

12. Coughlin, J. B.; Grosskreutz, D. C., and Stephen, C. R.: Transtracheal Analgesia, Southern M. J. 50:508 (April) 1957.

13. Gonzalo, P. H.: Potentiated Anesthesia Derived from Artificial Hibernation, A. M. A. Arch. Otolaryng. 65:13 (Jan.) 1957.

14. Altman, M. M., and Fialkov, G.: The Use of Meperidine in Peroral Endoscopies, A. M. A. Arch. Otolaryng. 65:221 (March) 1957.

15. Johnston, D. W.; Jensen, W. B., and Bird, M. L.: Successful Cardiac Resuscitation Following Tetracaine Reaction, A. M. A. Arch. Otolaryng. 66:592 (Nov.) 1957.

16. Holinger, P. H., and Johnston, K. C.: Clinical Aspects of Congenital Anomalies of the Trachea and Bronchi, Dis. Chest 31:613 (June) 1957.

17. Devenis, A. M., and Otis, R. D.: Tracheal Atresia Associated with Tracheo-Esophageal Fistula, J. Thoracic Surg. 34:405 (Sept.) 1957.

18. Kraus, M., and White, H.: Congenital Tracheo-Esophageal Fistula in the Neck Without Atresia, J. Pediat. 51:580 (Nov.) 1957.

19. Babbitt, D. P.: Double Tracheo-Esophageal Fistula Without Atresia. New England J. Med. 257:713 (Oct. 10) 1957.

20. Shaw, R. R., and Paulson, D. L.: Congenital Anomalies of the Esophagus, Am. J. Surg. 93:196 (Feb.) 1957.

21. Schaff, B., and Baum, G.: The Tracheal Bronchus, J. Thoracic Surg. 33:282 (Feb.) 1957.

22. Nelson, A. R.: Congenital True Esophageal Diverticulum, Ann. Surg. 145:258 (Feb.) 1957.

23. Maier, H. C.: Intramural Duplication of the Esophagus, Ann. Surg. 145:395 (March) 1957.

24. Netterville, R. E.: Agenesis of the Lung, Dis. Chest 31:457 (April) 1957.

25. Sullivan, H. J.: Supernumerary Diaphragm with Agenesis of Upper Lobe, J. Thoracic Surg. 34:544 (Oct.) 1957.

26. Nicks, R.: Agenesis of the Lung with Persistent Ductus Arteriosus, Thorax 12:140 (June) 1957.

27. Goldman, L., and Wilson, J. G.: Tracheal Diverticulosis. A. M. A. Arch. Otolaryng. 65:554 (June) 1957.

28. Beskin, C. A.: Rupture Separation of the Cervical Trachea Following a Closed Chest Injury, J. Thoracic Surg. 34:392 (Sept.) 1957.

29. Cummins, C. F. A., and Waterman, D. H.: The Management of Tracheal Stenosis, with a Case Report, Dis. Chest 31:375 (April) 1957.

30. Greer, A. E., and Winn, G. L.: Leiomyoma of Trachea, J. Thoracic Surg. 33:237 (Feb.) 1957.

31. Gibbs, N. M.; Taylor, M., and Young, A.: Von Recklinghausen's Disease in the Larynx and Trachea of an Infant, J. Laryng. & Otol. 71:626 (Sept.) 1957.

32. Barkley, R. S.; McSwan, N., and Welch, T. M.: Tracheal Reconstruction Without the Use of Grafts, Thorax 12:177 (Sept.) 1957.

33. Coleman, F. T.: Acquired Non-Malignant Esophago-Respiratory Fistula, Am. J. Surg. 93:321 (Feb.) 1957.

34. Bovornkitti, S., and Zabriskie, J.: A Technique of Bronchography in Children, with Evaluation of Contrast Media, Dis. Chest 32:388 (Oct.) 1957.

35. Björk, L., and Lodin, H.: Pulmonary Changes Following Bronchography with Dionosil Oily, Acta radiol. 47:177 (March) 1957.

36. Pesiri, E. J., and Sennott, W. M.: A Technique of Bronchography with Visciodol, Dis. Chest 31:548 (May) 1957.

37. Haidak, G. L.; Caplan, B., and Maloney, W. H.: Air Contrast Bronchography, J. Thoracic Surg. 34:63 (July) 1957.

38. Copp, S. E.; Krzyski, T. K., and Shane, S. J.: Experiences with Bronchography in the Management of Pulmonary Tuberculosis, Canad. M. A. J. 76:947 (June 1) 1957.

39. Autio, V.: Bronchospirometric Studies Relating to a Radiographic Method for Determining Differential Vital Capacity, Acta med. scandinav. Supp. 329, (Feb.) 1957.

40. Swenson, E. W., and Birath, G.: Resistance to Air Flow in Bronchospirometric Catheters, J. Thoracic Surg. 33:275 (Feb.) 1957.

41. Birath, G.; Bergh, N. P., and Swenson, E. W.: Bronchospirometric Investigations Before and After Segmental Resection and Lobectomy for Pulmonary Tuberculosis, Am. Rev. Tuberc. 75:710 (May) 1957.

42. Birath, G.; Swenson, E. W.; Ander, L., and Bergh, N. P.: The Definitive Functional Results after Partial Pulmonary Resection, Am. Rev. Tuberc. 76:983 (Dec.) 1957.

43. Birath, G., and Soderholm, B.: Bronchospirometric Investigations Before and After a Small Thoracoplasty, Am. Rev. Tuberc. 75:724 (May) 1957.

44. Watson, T. R., Jr.; Tyson, M. D.; Heller, M. L.; Cincotti, J. J., and Gaensler, E. A.: Bronchospirometry, Am. Rev. Tuberc. 75:730 (May) 1957.

45. Viikari, S. J., and Autio, V.: Bronchospirometry During Exercise in Sitting Position, Acta med. scandinav. 157:61 (March) 1957.

46. DeKornfeld, T. J., and Siebecker, K. L.: Changes in Oxygen Saturation During Bronchoscopies, Anesthesiology 18:466 (May-June) 1957.

47. Rivkin, L. M.; Read, R. C.; Lilliehei, C. W., and Varco, R. L.: Massive Atelectasis of the Left Lung in Children with Congenital Heart Disease, J. Thoracic Surg. 34:116 (July) 1957.

48. Shaw, R. R.; Paulson, D. L., and Kee, J. L., Jr.: Mucoid Impaction of the Bronchi, Am. Rev. Tuberc. 76:970 (Dec.) 1957.

49. Linton, J. S. A.: Long Standing Intrabronchial Foreign Bodies, Thorax 12:164 (June) 1957.

50. Semple, T., and Page, W. J. O.: Bilateral Bronchial Foreign Bodies, Lancet 1:769 (April 13) 1957.

51. deRoos, T.: Foreign Body in the Right Main Bronchus for 1½ Years, Canad. M. A. J. 77:960 (Nov. 15) 1957.

52. Palva, T.; Elo, R., and Saloheimo, M.: The Role of Bronchoscopic Studies in Pulmonary Tuberculosis, Dis. Chest 32:56 (July) 1957.

53. Hulse, W. F.: Bronchoscopy in Tuberculosis, A. M. A. Arch. Otolaryng. 65:487 (May) 1957.

54. O'Keefe, J. J.: The Significance of Endobronchial Lesions in the Management of Pulmonary Tuberculosis, Ann. Otol. Rhin. & Laryng. 66:1034 (Dec.) 1957.

55. Grosz, H. J.: A Case Study of Perforations of Tuberculous Lymph Nodes into Bronchi and Their Sequelae, Dis. Chest 32:690 (Dec.) 1957.

56. Kalbian, V. Z.: Bronchial Involvement in Pulmonary Sarcoidosis, Thorax 12:18 (March) 1957.

57. Citron, K. M., and Scadding, J. G.: Stenosing Non-Caseating Tuberculosis (Sarcoidosis) of the Bronchi, Thorax 12:10 (March) 1957.

58. Honey, M., and Jepson, E.: Multiple Bronchostenosis Due to Sarcoidosis, Brit. M. J. 2:1330 (Dec. 7) 1957.

59. Stevenson, J. G., and Reid, J. M.: Broncho-Pulmonary Aspergillosis, Brit. M. J. 1:985 (April 27) 1957.

60. Polk, J. W.; Cubiles, J. A., and Buckingham, W. W.: The Surgical Treatment of Chronic Progressive Pulmonary Histoplasmosis, J. Thoracic Surg. 34:323 (Sept.) 1957.

61. Peterson, P. A., Jr.; Soule, E. H., and Bernatz, P. E.: Benign Granular Cell Myoblastoma of the Bronchus, J. Thoracic Surg. 34:95 (July) 1957.

62. Ochsner, S.; LeJeune, F. E., and Ochsner, A.: Lipoma of the Bronchus, J. Thoracic Surg. 33:371 (March) 1957.

63. Fraser, K.: Bronchial Adenoma, Brit. J. Surg. 44:570 (May) 1957.

64. Overholt, R. H.; Bougas, J. A., and Morse, D. P.: Bronchial Adenoma, Am. Rev. Tuberc. 75:865 (June) 1957.

65. Ochsner, S., and Ochsner, A.: Bronchial Adenomas, Southern M. J. 50:1089 (Sept.) 1957.

66. Thompson, J. R.; Bosman, R. I., and Dumm, J. B.: Metastasizing Bronchial Adenoma with Heteroplastic Bone Formation, Dis. Chest 32:463 (Oct.) 1957.

67. Auerbach, O.; Gere, J. B.; Pawlowski, J. M.; Muehsam, G. E.; Smolin, H. J., and Stout, A. P.: Carcinoma in Situ: An Early Invasive Carcinoma Occurring in the Tracheobronchial Tree in Cases of Bronchial Carcinoma, J. Thoracic Surg. 34:298 (Sept.) 1957.

68. Valentine, E. H.: Squamous Metaplasia of the Bronchus, Cancer 10:272 (March-April) 1957.

69. Steele, C. H.: Bronchogenic Carcinoma, Laryngoscope 67:137 (Feb.) 1957.

70. Umiker, W. O.; DeWeese, M. S., and Lawrence, G. H.: Diagnosis of Lung Cancer by Bronchoscopic Biopsy, Scalene Lymph Node Biopsy and Cytologic Smears, Surgery 41:705 (May) 1957.

71. Foxen, E. H. M.: Ectopic Gastric Mucosa in the Cervical Esophagus, a Possible Cause of Dysphagia, J. Laryng. & Otol. 71:419 (June) 1957.

72. Smythe, C. M.; Osborne, M. P.; Zamcheck, N.; Richards, W. A., and Madison, W. M., Jr.: Bleeding from the Upper Gastro-Intestinal Tract, New England J. Med. 256:441 (March 7) 1957.

73. Vivas, J. R., and Palmer, E. D.: Incidence of Chronic Pulmonary Disease among Patients with Esophageal Disorders, Dis. Chest 31:568 (May) 1957.

74. Jackson, C. L.: Foreign Bodies in the Esophagus, Am. J. Surg. 93:308 (Feb.) 1957.

75. Zoller, H., and Bowie, E. R.: Foreign Bodies of Food Passages Versus Calcification of Laryngeal Cartilages, A. M. A. Arch. Otolaryng. 65:474 (May) 1957.

76. Perrone, J. A., and Cipcic, J. A.: Esophagoscopy for Removal of Foreign Body in a 7-Month-Old Premature Infant, A. M. A. Arch. Otolaryng. 65:521 (May) 1957.

77. Nichamin, S. J., and Gonne, W. S.: Foreign Body in the Esophagus of a Young Infant, J. Pediat. 51:197 (Aug.) 1957.

78. Peeler, M. B., and Riley, H. D., Jr.: Cardiac Tamponade Due to Swallowed Foreign Body, A. M. A. J. Dis. Child. 93:308 (March) 1957.

79. Sanderson, B. A.: Removal of Esophageal Foreign Bodies by the External Approach, A. M. A. Arch. Otolaryng. 66:305 (Sept.) 1957.

80. Equen, M.; Roach, G.; Brown, R., and Bennett, T.: Magnetic Removal of Foreign Bodies from the Esophagus, Stomach, and Duodenum. A. M. A. Arch. Otolaryng. 66:698 (Dec.) 1957.

81. Mustard, R. A.: Diverticula of the Esophagus. Canadian M. A. J. 76:822 (May) 1957.

82. McBurney, R. P.: Esophageal Diverticula, South. M. J. 51:45 (Feb.) 1957.

83. Lodge, W. O.: Excision and Pari-Passu Repair of Pharyngeal Diverticula, A. M. A. Arch. Otolaryng. 66:707 (Dec.) 1957.

84. Webster, B. H.: Spontaneous Perforation of an Oesophageal Diverticulum, Dis. Chest 31: 345 (March) 1957.

85. Atkinson, M.; Kramer, P.; Wyman, S. M., and Ingelfinger, F. J.: The Dynamics of Swallowing, J. Clin. Invest. 36:581 (April) 1957.

86. Cross, F. S.; Kay, E. B., and Johnson, G. F.: Studies on Neuromuscular Imbalance of the Esophagus, A. M. A. Arch. Surg. 75:631 (Oct.) 1957.

87. Kramer, P.; Atkinson, M.; Wyman, S. M., and Ingelfinger, F. J.: The Dynamics of Swallowing, J. Clin. Invest. 36:589 (April) 1957.

88. Cross, F. S.; Kay, E. B., and Johnson, G. F.: Neuro-Muscular Imbalance of the Esophagus Associated with Hiatal Hernia as Studied by Means of Cinefluorography and Intraluminal Pressure Recordings, J. Thoracic Surg. 34:736 (Dec.) 1957.

89. Ardran, G. M.; Kemp, F. H., and Wegelius, C.: Swallowing Defects after Poliomyelitis, Brit. J. Radiol. 30:169 (April) 1957.

90. Kaplan, S.: Paralysis of the Swallowing Mechanism Following Bulbar Poliomyelitis, A. M. A. Arch. Otolaryng. 65:495 (May) 1957.

91. Bugden, W. F.: Lower Esophageal Web, Am. J. Surg. 93:248 (Feb.) 1957.

92. Waldman, H. K., and Turnbull, A.: Esophageal Webs, Am. J. Roentgenol. 78:567 (Oct.) 1957.

93. Mathewson, C., Jr.; Schaupp, W. C.; Diamond, F. C, and French, S. W., III: Traumatic Rupture of the Esophagus, Am. J. Surg. 93:616 (April) 1957.

94. Chamberlain, J. M., and Byerly, W. G.: Rupture of the Esophagus, Am. J. Surg. 93:271 (Feb.) 1957.

95. Blalock, J.: Primary Esophago-Gastrectomy for Instrumental Perforation of the Esophagus, Am. J. Surg. 94:393 (Sept.) 1957.

96. Brewster, E. S.: Traumatic Perforation of the Esophagus Caused by Self Catheterization with Heavy Electric Wire, Am. J. Surg. 93:1021 (June) 1957.

97. Anderson, R. L.: Spontaneous Rupture of the Esophagus, Am. J. Surg. 93:282 (Feb.) 1957.

98. Naclerio, E. A.: The V Sign in the Diagnosis of Spontaneous Rupture of the Esophagus, Am. J. Surg. 93:291 (Feb.) 1957.

99. Benedict, E. B., and Nardi, G. L.: Peptic Stenosis of Esophagus, Am. J. Surg. 93:238 (Feb.) 1957.

100. Hale, H. W., Jr., and Drapanas, T.: Reflux Esophagitis, Am. J. Surg. 93:228 (Feb.) 1957.

101. Palmer, E. D.: Esophageal Ulcer in the Adult, A. M. A. Arch. Int. Med. 99:695 (May) 1957.

102. Som, M. L.; Shapiro, J. H., and Jacobson, H. G.: The "Gastric Lined" Esophagus with Esophagitis, Radiology 69:856 (Dec.) 1957.

103. Mustard, R. A.: Reflux Esophagitis, Canad. M. A. J. 76:811 (May 15) 1957.

104. Daly, J. F., and Cardona, J. C.: Corrosive Esophagitis, Am. J. Surg. 93:242 (Feb.) 1957.

105. Tatelman, M., and Burton, I. F.: Gastroesophageal Incompetence, Gastroenterology 33:991 (Dec.) 1957.

106. Stensrud, N.: Incompetence of the Cardia, J. Thoracic Surg. 33:749 (June) 1957.

107. Davis, E. D. D.: The Mechanism of the Cardiac End of the Esophagus, J. Laryng. & Otol. 71:471 (July) 1957.

108. Lindskog, G. E., and Kline, J. L.: The Problem of Hiatus Hernia Complicated by Peptic Esophagitis, New England J. Med. 257:110 (July 18) 1957.

109. Reid, W. O., and Robinson, A. W.: Hiatus Herniation, Gastric Mucosa Lining the Lower Esophagus, and Reflux Esophagitis Above, J. Laryng. & Otol. 71:760 (Nov.) 1957.

110. Marchand, P.: A Study of the Forces Productive of Gastroesophageal Regurgitation and Herniation Through the Diaphragmatic Hiatus, Thorax 12:189 (Sept.) 1957.

111. Atkinson, M.; Edwards, D. A. W.; Honour, A. J., and Rowlands, E. N.: The Esophagogastric Sphincter in Hiatus Hernia, Lancet 2:1138 (Dec. 7) 1957.

112. Barrett, N. R.: The Lower Esophagus Lined by Columnar Epithelium, Surgery 41:881 (June) 1957.

113. Engberg, H.; Thomsen, G., and Vesterdal, J.: Hiatus Hernia in Children, Acta pædiat. 46:371 (July) 1957.

114. Kelly, H. D. B.: Hiatal Hernia and Esophagitis, J. Laryng. & Otol. 71:386 (June) 1957.

115. Buchanan, A. C.; Bowers, W. F., and Sullivan, B. H., Jr.: Esophageal Hiatus Hernia, A. M. A. Arch. Surg. 74:276 (Feb.) 1957.

116. Fisher, H. C., and Johnson, M. E.: Esophageal Hiatal Hernia a Manifestation of Peptic Esophagitis, A. M. A. Arch. Surg. 75:660 (Oct.) 1957.

117. MacDougall, J. T., and Abbott, A. C.: Sliding Hiatus Hernia, Canad. M. A. J. 77:752 (Oct. 15) 1957.

118. Phear, D.: Hiatus Hernia, Lancet 1:708 (April 6) 1957.

119. Maurer, E. R., and Keirle, A. M.: The Serious Significance of Hiatus Hernia, A. M. A. Arch. Surg. 75:647 (Oct.) 1957.

120. Friedman, A. I.: Carcinoma of the Stomach in Association with Hiatus Hernia, Ann. Int. Med. 47:812 (Oct.) 1957.

121. Euphrat, E. J., and Delmonico, J. E., Jr.: Coexistent Achalasia of the Esophagus and Hiatal Hernia of the Cardiac End of the Stomach, J. Thoracic Surg. 34:395 (Sept.) 1957.

122. Olsen, A. M.; Schlegel, J. F.; Creamer, B., and Ellis, F. H., Jr.: Esophageal Motility in Achalasia after Treatment, J. Thoracic Surg. 34: 615 (Nov.) 1957.

123. Creamer, B.; Olsen, A. M., and Code, C. F.: The Esophageal Sphincters in Achalasia of the Cardia, Gastroenterology 33:293 (Aug.) 1957.

124. Olsen, A. M., and Creamer, B.: Studies of Esophageal Motility with Special Reference to the Differential Diagnosis of Diffuse Spasm and Achalasia, Thorax 12:279 (Dec.) 1957.

125. Traunce, J. R.; Deuchar, D. C.; Kauntze, R, and Thomas, G. A.: Studies in Achalasia of the Cardia, Quart. J. Med. 26:433 (Oct.) 1957.

126. Olsen, A. M.; Ellis, F. H., Jr., and Creamer, B.: Cardiospasm, Am. J. Surg. 93:299 (Feb.) 1957.

127. Moser, K. M., and McQuiston, C. F.: Achalasia, Am. Rev. Tuberc. 76:480 (Sept.) 1957.

128. Palmer, E. D.: On the Natural History of Esophageal Varices Which are Secondary to Portal Cirrhosis, Ann. Int. Med. 47:18 (July) 1957.

129. Palmer, E. D.: The Fate of Esophageal Varices in Cirrhosis Following Surgical Portal Decompression, Gastroenterology 32:861 (May) 1957.

130. Boyd, D. P., and Hill, L. D., III: Pharynx Tumors and Cysts of the Esophagus, Am. J. Surg. 93:252 (Feb.) 1957.

131. Ochsner, S., and Ochsner, A.: Lipoma of the Esophagus, Surgery 42:787 (Oct.) 1957.

132. Carter, M. G.: Endoscopic Intubation and Dilatation in the Management of Esophageal Carcinoma, Am. J. Surg. 93:266 (Feb.) 1957

133. Keeley, J. L.; Rooney, J. A.; Guzauskas, A. C., and Brynjolfsson, G.: Primary Malignant Melanoma of the Esophagus, Surgery 42:607 (Sept.) 1957.

Abstracts from Current Literature

Ear

MANAGEMENT OF THE INFERIOR CRUS IN OTOPLASTY. S. S. FEURERSTEIN, Eye, Ear, Nose & Throat Month. 37:385 (June) 1958.

Two patients with protruding ears were operated on by a technique for otoplasty which follows closely that of Becker. This is a meticulous surgical technique for reconstructing the antihelix. The correction of the protruding ear is achieved without causing the abnormal sharp ridges or projections which are usually present when cartilage is removed from the superior crus. Several modifications are offered to correct more satisfactorily those cases in which the outstanding deformity consists of absent inferior crura.

The cartilage incisions are similar to those of the Becker technique excepting in the area of the inferior crus. When the incision outlining the mesial boundary of the antihelix reaches the level of the inferior crus, it is carried through the inferior crus to the helical margin and then superiorly to connect with that portion of the helical incision which outlines the superior crus.

At the completion of the procedure the auricle should lie in a relatively normal position. The sutures are utilized to maintain the new position of the cartilaginous segments but not to overcome any unbroken spring of the cartilage. Skin and mattress sutures are removed after the seventh or eighth day.

The basic steps of the technique are demonstrated by line drawings, and the preoperative pathology and the postoperative results are illustrated by complete sets of photographs, including enlarged lateral views showing the contours of the auricular cartilage.

JENNES, Waterbury, Conn.

SURGICAL CORRECTION OF PROTRUDING EARS. J. B. ERICH, Eye, Ear, Nose & Throat Month. 37:390 (June) 1958.

Protrusion of the ears is corrected by a surgical procedure for otoplasty that is a modification of the early Luckett operation. A new antihelix is formed by making two parallel incisions 3 mm. apart through the full thickness of the cartilage in the proposed position for the antihelix. These incisions extend the full length of the ear up into the superior crus. The helix, however, is not divided directly above the superior crus. A horizoneal incision extends mesially to sever the continuity of the helix at the point where the helix joins the side of the head.

Mattress sutures are used to evert the cut edges of the cartilage and to produce a convex antihelix. The amount of correction of the ear is determined by the degree of tension placed on the mattress sutures. These mattress sutures, which are left in place for two weeks, may produce some ulceration of the underlying skin and may leave small scars. This undesirable complication can be avoided by inserting the mattress sutures so that they pass through the cartilage but not the skin on the lateral side of the parallel antihelical incisions, and through both cartilage and skin on the mesial side. The sutures may still produce some scarring on the mesial side, but since these scars are located on the concha they are invisible except on close inspection of the ear.

Before-and-after photographs are presented of two cases; these include anterior views of both cases and posterior views of one.

ABSTRACTER'S COMMENT: The photographic views shown are quite impressive. The final story in all methods of surgical correction of protruding ears, however, is the appearance of the contours of the surface of the auricle. Since this is demonstrated only by the lateral views, it would have been desirable to include such views in these cases.

JENNES, Waterbury, Conn.

ATTIC CHOLESTEATOSIS. A. TUMARKIN, J. Laryng. & Otol. 72:610-619 (Aug.) 1958.

The basic factors responsible for the development of cholesteatosis are presented by Tumarkin, who further says that each of these basic factors is a result, whether immediate or remote, of the diffuse upper respiratory disease of childhood. Six types of attic suppuration are considered and discussed in the management of these conditions. Whether to use a flap or not

to use a flap seems to be the question of the hour, and Tumarkin is dubious of the value of applying grafts. The management of each type of cholesteatosis is well presented.

LeJeune, New Orleans.

Results of Tympanoplasty: A Comparative Study of 76 Tympanoplasties, Contrasted with Modified Radical, and Radical Mastoidectomies. Bruce Proctor, Laryngoscope 68:888 (May) 1958.

The purpose of tympanoplasty is both to eradicate any diseased tissue and to preserve or improve hearing. Wullstein described five of these operations, each one applicable to a certain specific condition. Proctor evaluates 76 unselected, consecutive tympanoplasties of all types. They are compared with both radical and modified radical mastoidectomies. To be considered successful there had to be a hearing result of 30 db. or better. Three of four Type I tympanoplasties were successful. There were 23 Type II cases. Five cases showed gains to above 30 db. and seven who were above 30 db. to start with showed further gains. Six showed a loss. Of 26 Type III cases there was improvement in 20, decrease in 4, and no change in 2. Of 17 cases of Type IV, 2 showed some gain. Five out of six of Type V were unsatisfactory. However, these were done over old radical cavities.

Reasons for failures in all the types are discussed.

The results of tympanoplasty are compared with the modified radical operation. Seventy-five cases of the latter showed a result audiologically almost equal to the Type II tympanoplasty. The old radical operation (50 cases), however, was far inferior as to hearing improvement to Type III or IV tympanoplasty.

Hitschler, Philadelphia.

Report of a Recent Study on Dihydrostreptomycin Ototoxicity. Aram Glorig, Laryngoscope 68:1013 (June) 1958.

A very well controlled study is presented on the toxicity of dihydrostreptomycin and the pantothenic acid salt of dihydrostreptomycin. This latter was included because some evidence had been presented to show this drug to be less toxic. This study as well as others, however, does not confirm this. Both dihydrostreptomycin and its pantothenic acid salt do produce raised hearing thresholds. One thing to be noted is their toxicity. However, vestibular rather than cochlear harm is preferable, and therefore streptomycin is the preferable preparation, and it is equal therapeutically.

Hitschler, Philadelphia.

Office Closure of Tympanic Perforations: A New Approach. Arthur L. Juers, Laryngoscope 68:1207 (July) 1958.

Juers describes a method of closing tympanic perforations in the office.

The ear must be dry, free of infection or cholesteatoma, without cerumen, before any attempt at closure can be made. There must also be a marginal rim of drum present.

Under loupe magnification, the edges of the perforation are traumatized with special instruments (pictured). The epithelium is everted and the underlying substantia propria exposed, which starts the growth of granulations. These latter are then "rolled out" over the epithelium. Bonain's solution is used for anesthesia. Treatment is carried out at two-week intervals. A Cottonoid patch is placed over the perforation, moistened with a solution of urea and boric acid. This solution is used daily by the patient for its irritant (and hence stimulating) quality.

The results of this treatment are included. Ten central perforations were healed with an average of 4.2 treatments each.

Hitschler, Philadelphia.

Primary Closure of the Radical Mastoidectomy Wound: A Technique to Eliminate Postoperative Care. J. H. Thomas Rambo, Laryngoscope 68:1216 (July) 1958.

Rambo suggests a method for the rapid healing of the radical mastoid cavity. The simple mastoidectomy wound heals rapidly and completely. It is primary healing. A radical mastoid cavity can also be made to heal primarily, with all the advantages inherent in this kind of healing. Very little after-care is needed. The patient has no open cavity to suppurate or discharge. He may, for example, swim. This primary healing in radical mastoids is accomplished by filling the meticuously cleaned-out cavity with a pedicle flap of the temporal muscle. Its abundant blood supply keeps it viable. Skin flaps are prepared from the anterior and posterior canal walls. These are laid on the muscle. Complete healing results.

It must be noted that in cases where maximum hearing is desired, this method is not applicable. It is applicable where the bone conduction is already well down, the other ear is good, and where more elaborate procedures are not deemed advisable.

HITSCHLER, Philadelphia.

SYMPOSIUM: STAPES MOBILIZATION TWO YEARS LATER. H. P. HOUSE, Moderator, Laryngoscope 68:1389 (Aug.) 1958.

In this symposium the panel analyzes their thoughts regarding stapes mobilization today, as compared with a similar analysis by the same panel two years ago.

Dr. Murrell Lawrence this time is an added member, and he discusses the function of the middle ear. He explains what happens with and without fixation of the stapes with loss of ossicles or the drum membrane, and the function of two windows, including the expected hearing losses under these various situations. He also explains why the making of a very minute hole in the footplate of the stapes could cause a hearing gain. Almost every other facet of stapes mobilization is discussed by the various panel members.

It is emphasized that the operation is not a simple one, and that the hearing may become worse and that the patient should know this. Also that other complications may ensue.

Most of the members now have been attacking the footplate directly, with a larger percentage of initial success. However, the feeling was that with this footplate attack, regression was more frequent. It is evident that the operation is still in a state of flux, and will be so for some time in the future.

HITSCHLER, Philadelphia.

RESULTS OF FENESTRATION AFTER STAPES MOBILIZATION. ALLAN GOODMAN and THOMAS WALSH, Laryngoscope 68:1663 (Sept.) 1958.

Although several others have compared the results of fenestration without previous stapes mobilization with those in patients who had a previous unsuccessful stapes mobilization, there has been no statistical analysis. Here 38 cases of fenestration following a previous stapes mobilization are statistically compared with 227 cases of primary fenestration. Hearing level for speech (spondaic words) was the datum used and residual hearing instead of hearing gain as the measure of success. The results are that the two groups are not different, or, in other words, that the stapes mobilization procedure does not affect the results of the subsequent fenestration.

HITSCHLER, Philadelphia.

MOBILIZATION OF THE STAPES: HISTORICAL ASPECTS. JAMES A. MOORE, New York J. Med. 58:3095 (Oct. 1) 1958.

In 1876 Kessel in Germany removed the stapes, presumably to relieve otosclerosis. He was followed by L. Jack of Boston in 1892 and 1893, who reported his results as of "no value." Politzer's American edition, 1926, states that removal of the stapes is not only useless but may make the hearing worse! These failures may well be due to defective technique because of poor visualization. Now we have almost perfect illumination through the dissecting microscope ($\times$ 16), and a bloodless field can be obtained. The central area of the footplate has been fenestrated with "unbelievably good results—a gain of 32 db. . . ." In evaluation one must consider air- and bone-conduction together with speech reception. Four groups are studied: Group I has minimal stapes fixation. In 10 out of 12 cases the entire footplate was seen to be completely freed at operation. Group II includes more than 50 out of the 100 cases. Forty, or 74%, showed significant gains to 26 db. Fractured crura were common, but, even so, 32 cases improved to the 30 db. level. Group III, 27 cases, had some nerve degeneration; therefore the gains were limited to an average of 24 db. In Group IV there were seven cases in which nerve damage was considerable. They were not suitable for fenestration, but five showed a gain of 28 db. Only one patient improved to 30 db.

Complications were relatively few. However, the hearing may be worsened to a 10 db. loss! One must guard against injury to the facial nerve, total loss of residual hearing, otitis media, disabling vertigo or labyrinthitis. Fracture of either or both crura may occur, but seem not to be of great moment. The chorda were severed in five cases, three done deliberately to get better visualization. Few complained of any disturbance of taste—in fact they noticed more from an injured than from a cut nerve! Troublesome bleeding may occur in the peritympanic region after operation, but this can be largely prevented by care at operation. Small cotton balls soaked in tetracycline (Achromycin) solution or ointment may be applied.

With modern techniques mobilization of the stapes is giving increasingly good results to hearing. The variables are ascribed to the degree or amount of stapedial fixation and to how much the auditory nerve has degenerated. Some 90% of patients with normal bone-conduction and minimal air-conduction loss will be improved. And 30% of "borderline suitability will reach the useful 30 db. level." Even if mobilization fails, fenestration may still be tried if the patient will stand for it. Some 6% may show regression after mobilization, and in 3% the hearing may be worsened.

This paper was read before the Annual Meeting of the Medical Society of the State of New York on May 15, 1958. The discussion was led by Dr. Gordon G. Boyd, who said he favors the microscope to the loupe in obtaining visibility. Before doing anything to the stapes, he studies the layout critically and moves accordingly. He uses a needle explorer, and makes sure that the entire stapes moves, not merely a part of it. He adds to "complications" tinnitus, persistent perforation of the drum, adhesions, thickened membrane and facial deformity (wherein a suit for $250,000 was instigated in one case). Success depends largely upon experience in doing the operation frequently.

I. W. Voorhees, New York.

The Present Status of Stapedial Surgery. Samuel Rosen, New York J. Med. 58:3828 (Dec. 1) 1958.

In 1953 Rosen began to describe his results in releasing fixation of the stapes for the improvement of hearing in otosclerosis. Since then he has followed up the procedure year by year. One method was to make pressure directly against the neck of the stapes, thus indirectly loosening the footplate. The second manipulation was by working the footplate loose with a crochet-like sharp instrument, or making an opening in the footplate, thus exposing the vestibular membrane. Quite often one or both crura were fractured without moving the footplate; therefore with negative results. Now we have an improvement in most cases of 15 db. or even more. Advantages to the patient in these latter days are great: Hospital stay is short; there is brief stay-away time from work, no bandages, brief bed rest, no complications such as total hearing loss or facial paralysis. A review of some 3,000 cases gives courage to both patient and surgeon. The operation is "relatively simple." Compared with the difficult and laborious operation for establishing a new window, release of the footplate or removing a part of it seems now to be the operation of choice. But we still have to know in advance whether the auditory nerve is fairly intact, or at least not "degenerated."

This is No. 49 in the series "Recent Advances in Medicine" under the supervision of Robert Turell, M.D. Rosen works in Mount Sinai Hospital, New York City.

I. W. Voorhees, New York.

Sudden Deafness. D. Van Caneghem. Acta oto-rhino-laryng. belg. 12:5, 1958.

The author reviews the problem of sudden deafness. Excluding occlusion to the external canal and pathology of the middle ear, he presents 64 cases of sudden deafness due to either labyrinthine or retrolabyrinthine pathology. While these cases could not be verified by postmortem examination, the diagnosis was made purely on clinical manifestations. Acute intoxications, allergy, or acute viral infections were not factors in any of his cases. In 11 cases a diagnosis of acute edema was made. This was associated with an edema of the rhinopharynx, the external ear, or the general circulation. Intralabyrinthine hemorrhage was a possibility in two of his cases. He was not able to establish emboli as a cause in any of the cases.

However, a diagnosis of thrombosis or agglutination of the blood in the internal auditory artery or one of its branches was made in 45 cases. Arterial spasm can be primary but is usually secondary to a thrombosis or an agglutination.

He presented a number of case histories to illustrate each group.

Persky, Philadelphia.

Tympanoplasty and Chronic Hyperplastic Otitis Media. Mario Rius, An. oto-rino-laring. Uruguay 28:31, 1958.

Rius presents a new classification of chronic otitis media: 1. Tubal catarrh—a simple discharge coming through a central perforation of the tympanum, due to rhinopharyngeal pathology, allergy, or focal infection. Treatment is directed to the rhinopharynx. 2. Hyperplastic chronic otitis media. There is a mucoid or mucopurulent discharge, at times fetid, associated with a large central perforation and a marked impairment of hearing of the conductive type. There is a hyperplasia of the subepithelial layer of the mucous membrane of the middle

ear. Treatment is tympanoplasty plus local cortisone instillations and vaccine therapy. 3. Transforming chronic otitis media. This is characterized by a fetid mucopurulent discharge, a marginal perforation, and an involvement of the mastoid, either by osteitis or an eburnation of the mastoid cells. The mucous membrane may be polypoid in certain areas. Treatment is tympanoplasty plus cortisone and vaccine therapy. 4. Chronic suppurative otitis media with a secondary cholesteatoma. The treatment is tympanoplasty. 5. Chronic supurative otitis media due to cholesteatoma. Treatment is tympanoplasty. 6. Chronic suppurative otitis media associated with various types of specific processes or tumors of the middle ear. Treatment depends on the underlying pathology.

After discussing the pathology and symptomatology of chronic suppurative otitis media hyperplastica (Type 2), the author describes his modification of the tympanoplasty operation.

In conclusion, Rius stresses the importance of eliminating the active granulation and fibrous tissue. However, he emphasizes the importance of avoiding interference with free action of the tympanum, the ossicles, their muscles and their nutrient vessels. To prevent a recurrence of a regrowth of the granulation tissue, he advocates the use of the antibiotics, steroids, radiation and vaccine therapy.

PERSKY, Philadelphia.

THE CORTICOSTEROIDS IN THE TREATMENT OF MÉNIÈRE'S DISEASE. MARIO RIUS, An. oto-rino-laring. Uruguay *28*:52, 1958.

Rius uses a solution of the corticosteroids (2-benzyl-2-imidazoline [Priscoline] and hyaluronidase [Wydase]) for transtympanic injections in Ménière's disease. Its rationale is that there is a vasospasm of the intratympanic and labyrinthine vessels, which causes a faulty circulation in this area, or that there is an allergy, or an hormonal disequilibrium. There are a number of contraindications, such as acute infections, peptic ulcer, tuberculosis, chronic hepatitis, severe hypertension, diabetes, evidence of psychosis, pregnancy, and malignancy of the breast or the genitals.

The results have been equivocal. A fair number of cases have shown marked improvement, but his greatest failures have been in cases of hydrops of the cochlea. In spite of these questionable results, Rius believes that this method of treatment should not be abandoned, but should be used in some of the difficult cases that do not respond to other therapeutic measures.

PERSKY, Philadelphia.

FAILURES OF TYMPANOPLASTY AND THEIR CAUSES. R. MASPETIOL, Ann. oto-laryng. 75:480 (July-Aug.) 1958.

In a series of 400 cases of tympanoplasty Maspetiol has obtained most gratifying results in that the perforations remained closed and that there was a permanent improvement in hearing acuity. However, he has had a number of failures. These are due to (1) a persistence of the discharge; (2) either a partial or a total loss of the graft, and (3) a failure of improvement of hearing.

The persistence of the discharge is not always easy to localize. In chronic suppurative otitis, there may be an associated involvement of the bone, producing an osteitis. This may necessitate an antroatticotomy. Or there may be a latent mastoiditis, which would require a curettage of the mastoid. Again, the discharge may be of tubal origin. This willl require attention to the nose, sinuses, and rhinopharynx. Finally, the discharge may originate in the graft proper.

The failure of the graft may be due to partial or total necrosis, to adherence of the graft to the promontory, thus producing a loss of mobility of the tympanic membrane, and, finally, to a secondary perforation of the graft.

Lastly, there is a failure of improvement in the hearing acuity. This may be due to a fixation of the ossicular chain, or a destruction of part of the chain. There is also the element of faulty technique which must be considered.

Careful evaluation of each case of failure must be made, and often these can be corrected.

PERSKY, Philadelphia.

INDICATIONS AND RESULTS OF TRANSTYMPANIC INJECTIONS OF HYDROCORTISONE. R. GRIMAUD, and M. WAYOFF, J. franc. oto-rhino-laryng. 7:413 (May) 1958.

The authors recommend the transtympanic injection of hydrocortisone in noninflammatory otitis media, either in the acute phagocytic or in the chronic fibroblastic types. They use 1 to

1.5 cc. of the commercial hydrocortisone acetate, which is equivalent to 25 to 40 mg. of hydrocortisone. One to three injections are given at weekly intervals. A local anesthetic is not necessary. In a great number of cases, one treatment was sufficient, particularly in the acute and subacute cases. In otosclerosis and in sclerosing tympano-labyrinthitis of senility this treatment has very little merit.

In a group of 29 children, there were 20 with a sclerosing otitis. Nine had a complete cure, without any recurrence after two years. The other nine cases were adhesive otitis; there were three cures, one improvement, and five failures.

In 31 adults, 10 had frank fluid in the middle ear. All were cured with one injection. Fourteen cases had a dry tubal catarrh, either recent or recurrent. There were nine cures, one developed suppuration, and four were lost track of. In addition, there were five cases of old adhesive otitis, with two cures and three improvement in hearing acuity.

The results are spectacular in recent nonsuppurative inflammatory lesions. The improvement in hearing is always parallel to the reestablishment of the potency of the Eustachian tube. The authors also found a marked improvement in a number of cases of perceptive deafness.

PERSKY, Philadelphia.

CEREBRAL CEREBELLAR ABSCESSES OF OTITIC ORIGIN. I. TZETZU, M. CIOBANU, V. DIMITRIU, and S. MICLESCO, Rev. laryng. 79:449 (July-Aug.) 1958.

The authors present 13 cases of intracranial complications of otitic origin that were seen at the O. L. R. Clinic of Bucharest from January, 1952, to December, 1956. There were seven cures and six fatalities. The abscesses were localized in the temporosphenoidal region in seven cases and in the cerebellum in six cases.

The otitic pathology revealed a mesotympanic cholesteatoma in eight cases, a polyposis with a necrosing osteitis in five cases. The route of invasion to the intracranial structures was either by an osteitis of the antrotympanic tegmen, through the labyrinth, or from the lateral sinus. The labyrinthine route usually predisposes to cerebellar suppurations. This is also true of lateral sinus infections. In 12 of the 13 cases there was an associated meningitis. This was usually septic and purulent, but it may be aseptic (two cases) or aseptic serous (two cases). The organisms isolated were either Staphylococcus, Meningococcus, B. coli, or mixed.

The temporosphenoidal abscesses were usually encapsulated, but this was rarely found in the cerebellar abscess. The metastatic abscesses (vascular) did not have a capsule, irrespective of their location.

The symptomatology included fever, chills, headache, vomiting, convulsions, and coma. The diagnosis was difficult in a number of cases because of either (1) a lack of sufficient correlation between the symptoms and the otorrhea, or (2) the patient was admitted in coma. In spite of an evolution of an abscess, there may be a normal or subnormal temperature and a normal pulse. A bradycardia should lead one to suspect an intracranial lesion. Furthermore, other symptoms were not always present—such as projectile vomiting, ocular paralysis, or papilledema.

Treatment is complex. It includes surgical drainage, antibiotic therapy, cardia support, and cortisone therapy. The prophylactic care of an acute exacerbation of a chronic otorrhea is most important to prevent a progression to an intracranial complication. When this occurs, consultation with the neurologist, ophthalmologist, neurosurgeon, and internist may be necessary not only to help establish a correct diagnosis, but to assist in the proper management of the cases.

PERSKY, Philadelphia.

Larynx

THE USE OF ANTIBIOTIC-STEROID COMBINATIONS AS NEBULIZING AGENTS IN THE TREATMENT OF LARYNGEAL CONDITIONS: A PRELIMINARY REPORT. BERNARD L. SILVERBLATT, Laryngoscope 68:1081 (June) 1958.

Cortisporin is used in a No. 40 DeVilbiss nebulizer for home use. There are six to eight inhalations four times daily to begin with; then these are decreased as the symptoms improve.

Very good results are claimed in acute laryngitis, contact ulcer, laryngeal polyps (primarily and postoperatively), postoperatively in papillomas to prevent recurrences, and in hyperkeratosis and leukoplakia (primarily and postoperatively, and postoperatively) after the removal of tumors.

HITSCHLER, Philadelphia.

TRACHEAL FENESTRATION AS A NEW METHOD OF TREATMENT FOR ADVANCED EMPHYSEMA. E. E. ROCKEY, S. A. THOMPSON, C. F. BLAZSIK, and R. J. AHN, New York J. Med. 58:3607 (Nov. 15) 1958.

The authors aim to relieve not only emphysema, but also chronic bronchitis and certain cases of advanced pulmonary tuberculosis by making a permanent tracheocutaneous fistula. Experimental research began in 1955, and on Jan. 30, 1956, the first human tracheal fenestration was done at Metropolitan Hospital, New York. The operative technique cannot be described here save to say that it depends upon making two flap valves which are intended to be in apposition at all times save when a catheter is introduced for aspiration of secretions. This the patient can be taught to do at home and with relief to breathing. Four cases are described in detail. Indications are as follows: (1) advanced emphysema with or without pus, but with marked disability in breathing; (2) excessive suppuration in the lungs where surgical excision is not advisable; (3) fibrocystic disease of the pancreas with much suppuration in the lungs; (4) far-advanced pulmonary tuberculosis not subject to other therapy; (5) bulbar poliomyelitis with irreversible changes requiring a mechanical respirator. There are eight drawings, photographs, and x-ray pictures with four references to the literature. Emphasis is on the *newness* of the procedure. Seemingly some eight cases have been operated upon so far.

ABSTRACTER'S COMMENT: This is, of course, merely a palliative invention. It may save a patient from "drowning in his own secretions. Nothing is said of cultures, vaccines or medication as adjuvants. Should one be content with removal of secretions only, and not attempt to change the quality or quantity of the secretions? The doughy, sticky secretions must be difficult to remove by suction in many instances.

I. W. VOORHEES, New York

Nose

MANAGEMENT OF NASAL FRACTURES: AN EVALUATION. I. B. GOLDMAN, Eye, Ear, Nose & Throat Month. 37:405 (June); 38:464 (July) 1958.

It is important to survey carefully the injury and to determine the degree, extent, and severity of the nasal fracture. Nasal fractures are easily identified by the visible and palpable signs of the injury. Frequently, however, because of the absence of pain and slight bleeding there is a tendency not to intervene but to go through a period of so-called "watchful waiting." By the time the nose becomes obviously crooked when the swelling subsides, a simple reduction may or may not be feasible. A nasal fracture is frequently missed because of a lack of recognition of the latent signs of a fracture, the desire not to subject the patient to operation, and the dependence upon x-ray evidence of fracture. Physicians may neglect to correct a clinically deviated nose if they depend upon the validity of a report, "Fracture in good position." It is also important from the therapeutic as well as from the medicolegal point of view to ascertain whether there has been a preexisting deformity.

The majority of nasal fractures can be corrected by closed reduction. Open reduction is required in delayed reduction or in incorrectly managed fractures. The rhinoplastic approach is indicated in the injured patient who has had a previous nasal deformity involving the external nose or the septum. A rhinoplasty should be performed in cases of nasal fractures wherein the caudal end of the septum has been injured and displaced or when the columella is deformed because of a fracture or dislocation of the anterior nasal spine.

JENNES, Waterbury, Conn.

VOICE QUALITY AFTER ADENOTONSILLECTOMY. C. N. HANLEY and C. C. MANNING, J. Speech & Hearing Disorders 23:257 (Aug.) 1958.

Thirty-seven children coming to the King County, Washington, Health Department for adenoidectomy were recorded preoperatively and for a period of 37 days postoperatively. The entire series was randomized and presentd in two different ways to a group of expert judges for scaling. There were significant mean differences betwen pre- and postoperative recordings in the direction of great hypernasality in postoperative voices. No significant differences were found in means of postoperative recordings. A significant difference was determined between reversed and normal play-back techniques, with reversed play-back procedures resulting consistently in higher scale values.

PALMER, Wichita, Kan.

TREATMENT OF FRACTURES OF THE FRONTAL AND ETHMOID SINUSES. HOLLIE E. MCHUGH, Laryngoscope 68:1616 (Sept.) 1958.

The various types of fractures involving the frontal and ethmoidal sinuses are described. Intracranial complications are frequent and often fatal, particularly if proper surgical management is not instituted. The chief danger of fracture of the frontal and especially the ethmoidal sinuses is laceration of the dura and spread of the infection intracranially. In frontal sinus fractures there may be abscess or aerocele formation instead. The sure sign of a dural tear is leakage of cerebral spinal fluid or the presence of an aerocele.

Treatment should be in conjunction with a neurosurgeon. Simple uncomplicated fractures of nondiseased sinuses may require no treatment whatsoever, but, if the sinuses are diseased or there is a dural tear, danger of meningitis exists and surgery is indicated. This may be a mere trephine opening or extensive bone removal, depending, of course, on the severity of the lesion.

HITSCHLER, Philadelphia.

CLINICAL AND THERAPEUTIC CONSIDERATIONS OF BUCCAL-SINUS FISTULAS. A. J. M. ROCA DE VINALS and R. VERGAS LLARDENT, Acta oto-rhino-laring. iber-am. 9:284, 1958.

The authors discuss the various relationships between the premolar teeth and the nasal fossa and the maxillary antrum. It is because of the many anatomic variations of the superior maxilla that there occur many of the fistulas following a dental extraction.

Treatment depends on the type, duration, and position of the fistula. A Caldwell-Luc operation is frequently necessary. In addition, the authors employ various types of flaps, but their preference is for the Ramadier modification. A large section of the palatal mucosa is elevated, care being taken that the superior palatine artery is included in the pedicle, and the flap is rotated to cover the fistula and attached to the gingival margin.

PERSKY, Philadelphia.

LATE RESULTS WITH ACRYLIC IMPLANTS IN OZENA. G. JANTSEK and A. GOYZE, Pract. oto-rhino-laryng. 20:224 (July-Sept.) 1958.

The authors review the early and late results of acrylic implants for ozena. They have made modifications in the original technique of Eyries. There are a number of precautions to observe. These are (1) case in the prevention of any septal perforations; (2) avoidance of occlusion either of the lacrymal duct or of the natural ostia of the sinuses; (3) insertion of the implants in a manner to avoid small pockets that may lead to subsequent suppuration and the extrusion of the implants, and (4) the insertion of enough implants to produce a narrowing of the nasal cavity, but not a complete occlusion.

An early evaluation of 90 cases showed excellent results in 26.6%, good in 52.3%, and unchanged in 21.1%. These results were evaluated on the basis of the following symptoms: headache, nasal obstruction, nasal discharge, crusting, and fetor.

However, when these cases were reviewed after six years, it was found that 61 cases expelled the implant at one time or another. The study showed that excellent results were obtained in 23%, good in 33.9%, and poor results in 43.1%.

In conclusion, the authors state that their technique is very simple, is well tolerated by the patient, does not have any prolonged postoperative treatment, and complications are rare.

PERSKY, Philadelphia.

Pharynx

NASOPHARYNGEAL FIBROMA. JAMES P. FRASER, J. Laryng. & Otol. 72:626 (Aug.) 1958.

The author considers nasopharyngeal fibromas as rare tumors found most frequently on the lateral wall of the nasopharynx above the Eustachian orifice with outstanding clinical features of nasal obstruction, epistaxis, and deafness. Various types and methods of treatment are discussed and considered, and three cases are reported. Of particular interest was the lateral rhinotomy approach and the surgical removal of the fibroma under induced hypothermia. The author reports considerably less bleeding during operation and much less postoperative shock. This hypothermia opens a new and apparently safer approach to these potentially hemorrhagic tumors.

LEJEUNE, New Orleans.

CANCER OF THE TONSIL. JOHN S. LEWIS, New York J. Med. 58:2094 (Sept. 15) 1958.

This was a "radio talk" over WNYC and is No. 12 in a series of Cancer Alerts under the aegis of the New York Academy of Medicine and New York Cancer Committee. Lewis says that chronic infection and constant irritation may "incite the development of cancer of the tonsil." Some 2% of human cancers occur in the tonsils, and it is the second site of origin of cancer of the throat. Older persons are most likely to have it. Males at 85% and females at 15% are the sufferers. Repeated attacks of infection are in the history. Poor dental and oral conditions are usually found. Pain may not be manifest for months after the growth is perceived, but earache is common. A lump at the angle of the jaw is an important feature and gives one an idea of extension as it becomes progressively larger. Dysphagia is not an early symptom. A biopsy is of prime importance. "Cancer of the tonsil can be cured by surgery, x-ray treatment or a combination of both. Many cases are being cured even though neck node metastases are present." Frequent inspections must be made to determine recurrences, if any.

ABSTRACTER'S COMMENT: This is a very good summation for public education purposes. One may be permitted a bit of hyperenthusiasm to allay fears and encourage hopefulness.... Sarcoma of the tonsils may be mistaken for "quinsy." I have seen such a case where "several incisions brought no pus." Since these tumors are usually encapsulated and progress rather slowly, they offer better chance of a "cure" than the basal-celled carcinoma.

I. W. VOORHEES, New York

HYPERTROPHY OF WALDEYER'S RING AND CHRONIC MAXILLARY SINUSITIS (LATENT) IN INFANTS. MARCEL VAN EYCK, Acta oto-rhino-laryng. belg. 12:39, 1958.

Van Eyck discusses the relation between chronic maxillary sinusitis and hypertrophy of Waldeyer's ring in infancy. He presents 68 cases from his private practice, of which 21 had a uni- and 47 had a bilateral sinusitis. These were verified by x-ray examination and antral lavage. Thirty-five of the infants had previous surgery—a tonsillectomy, adenoidectomy, or both. In most of these patients there was a recurrence of the vegetations. Allergy was an associated finding in 50% of his cases, but these cases usually did not respond to antiallergic treatment. He believes that the allergy is directly related to the infectious process in the sinus, and that cure is dependent upon eliminating the suppuration in the sinus. This may eliminate the necessity for surgery.

PERSKY, Philadelphia.

NEPHRITIS OF RHINO-PHARYNGEAL ORIGIN AND ITS TREATMENT. J. DESPONS and J. RENAULT, Rev. laryng. 79:466 (July-Aug.) 1958.

The authors distinguish three types of infectious nephritis—interstitial, tubulonephritis, and glomerulonephritis. Their discussion deals only with the latter group. They believe that there is a direct relationship between this group and rhinopharyngeal infections, be they acute or chronic.

There is the acute glomerulonephritis which is associated with an acute tonsillitis. The symptoms include albuminuria, cylinduria, hematuria, azotemia, edema, and occasionally cardiovascular syndromes. This stage usually responds fairly rapidly to proper therapeutic measures, which should include the removal of any infectious focus, particularly a tonsillectomy.

The chronic forms of nephritis present the usual symptoms of pallor, fatigue, anemia, emaciation, edema, and hypertension.

The commonest organism isolated from the urine is the Streptococcus A. There are several theories as to the route of involvement of the kidneys—bacteremic, vascular, toxinogenic, and allergic.

The medical treatment of the acute nephritis includes the antibiotics, vitamins P and C, the antihistamines, cortisone, and ACTH, plus a low-salt diet and a limitation of the fluid intake. The surgical treatment is tonsillectomy. Diathermic coagulation or roentgen therapy of the tonsils is not recommended. The response to surgery varies with the chronicity of the nephritis. In the early acute cases the urine may become normal within a month, while in the more chronic form—especially where the pathology may have become irreversible—improvement may not occur for four to six months.

PERSKY, Philadelphia.

Miscellaneous

DISABILITY OF SPEECH RESULTING FROM MALPOSITIONED CERVICAL SPINE FOLLOWING POLIOMYELITIS. WALLACE A. GOATES and JAMES F. BOSMA, J. Speech & Hearing Disorders 23:283 (Aug.) 1958.

Poliomyelitic sequelae resulting in cervical spinal lordosis and scoliosis have been observed to produce speech disorders, arising from lateral displacement, compression and immobilization of the larynx; distortion, displacement and compression with occlusion of the pharynx, fixation of the mandible and reduction in mobilization of articulatory mechanisms of speech. When the lordosis is relieved speech improves. Speech therapy without the freeing of these structures proved to be unrewarding.

PALMER, Wichita, Kan.

THE ROUTINE BLEEDING AND CLOTTING TIME TESTS: THEIR MEDICOLEGAL STATUS. RUSSELL FLETCHER, Laryngoscope 68:1087 (June) 1958.

Evidence is presented to show that the bleeding and coagulation tests, so often routinely performed prior to tonsillectomy, are not necessary. They often do not show a real bleeding tendency, including some of the most pronounced blood dyscrasias. On the other hand, they may give a false sense of security, which is undesirable. A good history and physical examination is far to be preferred, and it is much more reliable, to ask about subcutaneous bleeding and bruising.

Many outstanding hospitals and clinics have abandoned the routine tests. It is therefore considered justifiable not to use them and without fear of legal complications.

HITSCHLER, Philadelphia.

AN OBJECTIVE APPROACH TO THE COMPLAINT, LUMP IN THE THROAT. J. ARNOLD HENRY, Laryngoscope 68:1257 (July) 1958.

This article points out that the symptom "lump in the throat" is more likely to be organic than functional. In 175 cases, more than half had at least an organic background, and many were entirely organic.

Previous reports on this subject together with the present series illustrate the varied etiology.

Pharyngitis, particularly of the lingual tonsil, heads the list. There are also sinusitis with postnasal drip, old esophageal lesions, tumors, foreign bodies, cervical osteoarthritis, pharyngeal muscular spasm from various causes, and other definite lesions. These must be eliminated before a diagnosis of functional disease is made.

There must be a careful history, with a sympathetic approach (for many times there is at least an overlay of hysteria). Then a careful examination, keeping in mind the causes mentioned above. After this, if necessary, there should be a thorough x-ray study, including the chest, cervical spine, and sinuses. Barium studies are often desirable. Finally a direct esophageal examination may be made. Many lesions of this area produce the "lump in the throat" symptom.

Management of course, depends upon the cause, whether it be alleviation of the pharyngitis, sinusitis, removal of the foreign body, or dilation of the esophagus. The thought to keep in mind, however, is that this symptom, "lump in the throat" shouldn't be considered functional until all other causes are eliminated.

HITSCHLER, Philadelphia.

MEDICAL AND SURGICAL TREATMENT OF BELL'S PALSY. EDMOND P. FOWLER JR., Laryngoscope 68:1655 (Sept.) 1958.

Fowler discusses seven cases of Bell's palsy in which he operated. On splitting the nerve sheath he found the nerve to bulge out. Many have thought that there is pressure present and that it is this pressure which causes the paralysis. However, in animal experiments moderate pressure did not cause paralysis. Fowler believes the cause is probably ischemia and that the lack of blood in severe cases, decompression of the nerve should be done very early before degeneration can take place. His results have been uniformly excellent, with the function returning soon in every case.

With medical treatment, results vary. When procaine was used, 12 out of 14 cases had full improvement, and these on the average 12.3 days. Of the controls 72% had full improvement in an average 76.1 days. Results varied with cortisone and nicotinic acid, and physiotherapy. Probably the best results from medical treatment were obtained by the use of

nicotinic acid and intravenous procaine. However, it is emphasized that surgical decompression should be instituted if medical treatment does not produce improvement in a few days.

HITSCHLER, Philadelphia.

INCREASED RESISTANCE TO ANTIBIOTICS AMONG MICROORGANISMS IN A GENERAL HOSPITAL. ALFRED J. WEIL, New York J. Med. 58:3102 (Oct. 1) 1958.

From the laboratories of the Bronx General Hospital Weil gives the results of his studies during 1956-1957. There is a great amount of material in the literature covering the past seven years, and the general trend has been toward increasing resistance of bacteria to the antibiotics. For this study seven of the commoner germs have been taken by Dr. Weil, viz., Streptococcus, Staphylococcus, Escherichia, Aerobacter, Proteus, Pseudomonas aeruginosa, Alcaligenes faecalis. Since 1951 several articles have been written concerning infections in the wards, especially in re staphylococcic infections. Against these were the following: penicillin, erythromycin, tetracycline, chloramphenicol, streptomycin, and polymixin. The methods of testing are described in detail. Resistence was found of streptococci to tetracycline; of staphylococci to the same, and also to streptomycin, and, of to a lesser degree, to penicillin, erythromycin, and chloramphenicol; of Escherichia to tetracycline and polymixin B; of Aerobacter to tetracycline, chloramphenicol, and polymixin B, and, finally, of Bacillus faecalis alcaligenes to tetracycline and chloramphenicol. The statistics may vary as to such results in hospitals compared with those in doctors' offices. The increased resistance to tetracycline was impressive. Neomycin and chloramphenicol were seemingly more potent in multiple infections.

ABSTRACTER'S COMMENT: We should like to be specific if we could, that is, be able to pick out a definite antibiotic for a definite enemy. This is not yet possible. Trial and error must still be our method of procedure. . . . Here the danger is the time element; in severe infections, we may be unable to administer the most potent antibiotic soon enough to be effective.

I. W. VOORHEES, New York

ESOPHAGEAL HIATUS HERNIA: A SYMPOSIUM, New York J. Med. 58:3271 (Oct. 15) 1958.

A group, chiefly from the University and hospitals of Buffalo, discussed the esophagus at the Annual Meeting of the Medical Society of the State of New York, in New York City during May, 1958. The symposium had to do chiefly with hiatus hernia,—"the upside-down stomach" of the newspapers.

For the direct tube specialist the most interesting was, perhaps, the paper of Dr. Murray N. Anderson of Buffalo University School of Medicine and the E. J. Meyer Hospital. He said that direct visualization of the gullet differentiated simple spasm from stricture or cancer of the esophagus. It is not a procedure besought with great difficulties as many seem to think. "Extension of its use would be of great benefit" in diagnosis and in laying plans for therapy. In hernia of the esophagus, "it makes possible a more exact definition of the pathologic process, and is of distinct value in the choice of the most appropriate therapeutic program."

ABSTRACTER'S COMMENT: All of the authors whose articles appear in this issue of the *Journal* are authorities in their fields, and the numerous diagrams, figures, etc., make for an exhaustive study of this sometimes baffling condition. The entire group of papers is well worth reading.

I. W. VOORHEES, New York

MEDIASTINAL LYMPH NODE PUNCTURE THROUGH THE TRACHEAL CARINA. E. SCHIEPPATI, Surg. Gynec. & Obst. 107:243 (Aug.) 1958.

Puncture aspiration through a deformed carina is proposed as a supplemental endoscopic technique in order to obtain lymphatic material for histologic study. A 50 cm. needle of 1 mm. diameter, with mandrel and properly sterilized, is inserted through a bronchoscope into the carina. The mandrel is removed; a syringe is attached; the needle is advanced 2 or 3 cm., and aspiration is carried out. The entire apparatus is then carefully withdrawn, avoiding any aspiration of tracheal contents. Routine tracheal aspiration is then effected and 30% silver nitrate is applied to the puncture site for hemostasis. The specimen obtained is washed with saline into a formalin-containing test tube and prepared for pathological study in the usual manner.

Mediastinal invasion by bronchogenic or esophageal cancer can be effectively demonstrated by this method in an appreciable percentage of cases. The author states that the method is free of great risk and advocates its application to other diseases, such as mediastinal tumors,

Hodgkin's disease, sarcoidosis, etc. He cautions against its use in widespread soft tuberculous mediastinal disease.

FRIEDBERG, Chicago.

COMPLICATIONS OF LOCAL ANESTHESIA IN OTOLARYNGOLOGICAL PROCEDURES. E. BERINI GIMENEZ, Acta oto-rino-laring. iber-am. 9:169, 1958.

Untoward reactions to local anesthesia may occur at any time and in any type of patient. These may be due to the toxicity of the anesthetic or the individual intolerance of the patient. Among the various causes of death one can mention errors of technique (parenteral administration of cocaine), excessive dosage, lymphatic states, individual sensitivity, and psychic or emotional states. The symptomatology includes the nervous phenomena such as headache, convulsions (local and general), somnolence, coma, etc.; respiratory, cardiovascular, and neurovegetative symptoms. The pathology is not clear-cut, and no particular site or organ can be implicated in all cases.

Prophylaxis is important in that the patient is given the proper preoperative medication, the anesthetic is freshly made up, is used in the proper manner, minimal dosage employed, and that any evidence of intolerance or idiosyncrasy to the medication be immediately countered by the proper measures. This includes the barbiturates, cardiac and respiratory stimulants, vasodilators, etc.

The author presents four cases, three of whom died in spite of all heroic measures of treatment.

PERSKY, Philadelphia.

News and Comment

ANNOUNCEMENTS

Functional Otological Postgraduate Course in Microsurgery.—A practical course in surgical techniques will be conducted from July 1 to July 10, 1959, in English, by Michel Portmann, M.D., Associate Professor, at the University of Bordeaux Ear, Nose, and Throat Department (Prof. Georges Portmann, Director).

During this period, every assistant attending the course will be enabled to watch surgical interventions in the theater, to follow theoretical courses illustrated with films regarding the major techniques, and to work on his own daily anatomical specimen with all the requisite material, including the binocular microscope.

The number of assistants is limited to 10.

Fees: $100 (to be paid at the Faculty of Medicine in Bordeaux at the beginning of the course).

Applications should be sent to Dr. Michel Portmann, 45 Cours Foch, Bordeaux, France.

Pan-Pacific Surgical Association.—The Eighth Congress of the Pan-Pacific Surgical Association will be held in Honolulu, Sept. 28 through Oct. 5 in 1960.

All members of the profession are cordially invited to attend and are urged to make arrangements as soon as possible if they wish to be assured of adequate facilities.

An outstanding scientific program by leading surgeons promises to be of interest to all doctors. Nine surgical specialty sections are held simultaneously.

Further information and brochures may be obtained by writing to Dr. F. J. Pinkerton, Director General of the Pan-Pacific Surgical Association, Suite 230, Alexander Young Building, Honolulu 13, Hawaii.

Directory of Otolaryngological Societies *

FOREIGN AND INTERNATIONAL

Canadian Otolaryngological Society
(Société Canadienne d'Otolaryngologie)

President: Dr. Robert T. Hayes, 42 Cobourg St., St. John, N. B.
Secretary: Dr. Donald M. MacRae, 34 Spring Garden Rd., Halifax, N. S.

Venezuela Otorhinolaryngology Society

President: Dr. Alfredo Celis Pérez.
Secretary: Dr. Jesús Miralles.

International Association of Allergology

President: Dr. Samuel M. Feinberg, 303 E. Chicago Ave., Chicago 11.
Secretary-General: Dr. José Quintero Fossas, Paseo 313, Vedado, Havana, Cuba

International Bronchoesophagological Society

President: Dr. Jo Ono, Tokyo.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.

International College of Surgeons, Section of Ophthalmology and Otolaryngology

President: Dr. John R. Lindsay, 950 E. 59th St., Chicago
Secretary-Treasurer: Dr. Louis Savitt, 3403 W. Lawrence Ave., Chicago 25.
Meeting: 24th Annual Congress, Palmer House, Chicago, Sept. 14-17, 1959.

International Congress of Audiology

General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.
Place: Leyden, Netherlands.

International Course in Paedo-Audiology

General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.
Place: Groningen, Netherlands.

Mexican Society of Otolaryngology

President: Dr. Rafael Giorgana
Secretary: Dr. Carlos Valenzuela, Monterrey 47-201, Mexico 7, D. F.

Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. Paul H. Holinger, Chicago.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Victor R. Alfaro, Washington, D. C.
Secretary: Dr. Walter E. Heck, San Francisco.
Meeting: Atlantic City, June 8-12, 1959.

American Academy of Ophthalmology and Otolaryngology

President: Dr. LeRoy A. Schall, 243 Charles St., Boston.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

American Board of Otolaryngology

President: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse 10, N. Y.
Secretary-Treasurer: Dr. Dean M. Lierle, University Hospitals, Iowa City.

American Broncho-Esophagological Association

President: Dr. Francis W. Davison, Geisinger Memorial Hospital, Danville, Pa.
Secretary: Dr. F. Johnson Putney, 1712 Locust St., Philadelphia 3.

American Laryngological Association

President: Dr. Fred W. Dixon, 2060 E. 9th St., Cleveland.
Secretary: Dr. James H. Maxwell, University Hospital, Ann Arbor, Mich.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse 10, N. Y.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.

SECTIONS:

Eastern.—Chairman: Dr. Joseph L. Goldman, New York.
Southern.—Chairman: Dr. James T. King, Atlanta, Ga.
Middle.—Chairman: Dr. Alfred J. Cone, St. Louis, Mo.
Western.—Chairman: Dr. Robert C. McNaught, San Francisco, Calif.

American Rhinologic Society

President: Dr. Kenneth H. Hinderer, 402 Medical Arts Building, Pittsburgh 13.
Secretary: Dr. Robert M. Hansen, 1735 N. Wheeler Ave., Portland, Ore.
Annual Clinical Session: Chicago, October, 1959—Definite time and place to be annocunced.
Annual Meeting, Chicago, October, 1959—Definite time and place to be announced.

American Otological Society, Inc.

President: Dr. Moses H. Lurie, 483 Beacon St., Boston.
Secretary: Dr. Lawrence R. Boies, University Hospitals, Minneapolis 14.

American Otorhinologic Society for Plastic Surgery, Inc.

President: Dr. Armand L. Caron, 36 Pleasant St., Worcester, Mass.
Secretary: Dr. Joseph G. Gilbert, 75 Barberry Lane, Roslyn Heights, N. Y.

American Society of Facial Plastic Surgery

President: Dr. Trent W. Smith, 327 E. State St., Columbus 15, Ohio.
Secretary: Dr. Samuel M. Bloom, 123 E. 83rd St., New York 28.
Meeting: July 15, 1959, New York.

American Society of Ophthalmologic and Otolaryngologic Allergy

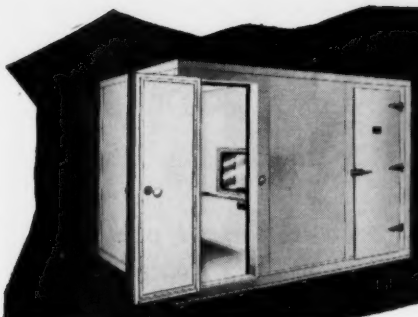
President: Dr. Joseph W. Hampsey, Grant Building, Pittsburgh 19.
Secretary-Treasurer: Dr. Daniel S. DeStio, 121 S. Highland Ave., Pittsburgh 6.

Otosclerosis Study Group

President: Dr. E. P. Fowler Jr., 180 Fort Washington Ave., New York 32.
Secretary-Treasurer: Dr. Arthur L. Juers, 1018 Brown Building, Louisville 2.

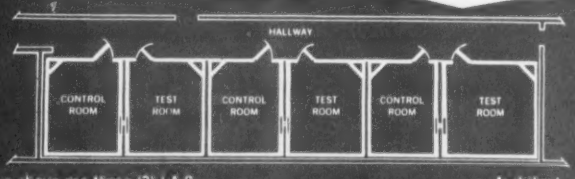
Society of Military Otolaryngologists

President: Lt. Col. Stanley H. Bear (MC), USAF, Maxwell AFB, Ala.
Secretary-Treasurer: Capt. Maurice Schiff (MC), U. S. N., U. S. Naval Hospital, Oakland, Calif.



IAC Series 400-CT standard Audiometric Examination Room combines an Examination Area and Control Area in one unit.

standard-ized...or...special-ized



Shown above are three (3) I.A.C. Series "400-CT" Rooms now in use in the New Office Building of Howard P. House, M.D., Los Angeles, Calif.

Architect:
Armand Monaco
Los Angeles, Calif.

IAC
Your Standard of Silence

audiometric examination rooms

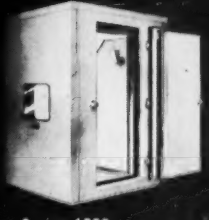
provide the ultimate in required performance at lowest cost!

IAC advanced acoustical engineering makes available to the Otolaryngologist and Audiologist, Audiometric Examination Rooms with performance guaranteed by known results in hundreds of existing installations.

IAC Audiometric Examination Rooms are prefabricated in many models to meet your acoustical and structural requirements. Where special audiometric installations are desired, IAC's engineering staff is at your, and your architect's disposal, for the design, construction and installation of complete Audiology Clinics and Research Centers – at the same low cost per square foot as the standard rooms!

Professional literature will be furnished on request.

Series 400



Series 1200

Medical Products Department

INDUSTRIAL ACOUSTICS COMPANY, INC.

341 Jackson Avenue • New York 54, N. Y.

Dept. O-5

ALCOHOLISM

an important problem in today's living!

The following articles from TODAY'S HEALTH are now available in one pamphlet for 50 cents . . .

ALCOHOLICS ANONYMOUS. Written from the standpoint of a member, the basic treatment procedures are described and the psychological problems confronting the alcoholic are discussed.

ALCOHOL AND CIRRHOSIS OF THE LIVER. Relationship between alcohol, diet and cirrhosis. Increasing stress on nutritional differences. by Russell S. Boles

HOW TO HELP A PROBLEM DRINKER. Understanding the alcoholic's capabilities, the necessity of help, causes of his condition. by Edward A. Strecker and Francis T. Chambers, Jr.

THE TREATMENT OF ALCOHOLISM. Tracing the steps from convincing the alcoholic that he is sick through treatment and cure. by Lewis Inman Sharp

CONDITIONED REFLEX TREATMENT OF CHRONIC ALCOHOLISM. Its place among methods of treatment today, its development and correlation with personality factors. by Walter L. Voegtlin

INSTITUTIONAL FACILITIES FOR THE TREATMENT OF ALCOHOLISM. Comparative differences, in drinking, with the last century, new establishments and methods of treatment, lack of trained personnel. by E. H. L. Corwin

other pamphlets available . . .

ALCOHOLISM IS A DISEASE. A discussion by the Chairman of the A.M.A.'s Committee on Alcoholism. by Marvin A. Block, M.D., 8 pages, 15 cents

I AM THE WIDOW OF AN ALCOHOLIC. Three articles combined, by Virginia Conroy, 16 pages, 20 cents

HOW EXPERTS MEASURE DRUNKENNESS. A partial transcript of an actual courtroom case. by H. A. Heise, 8 pages, 15 cents

BARBITURATES, BOOZE AND OBITUARIES. A discussion of the dangers of mixing alcohol and barbiturates. by Donald A. Dukelow, 4 pages, 10 cents

TWELVE STEPS FOR ALCOHOLICS. A frank discussion of the meaning of an alcoholic behavior. by Richard Lake, 6 pages, 10 cents

address requests to . . .

ORDER DEPARTMENT

AMERICAN MEDICAL ASSOCIATION
535 NORTH DEARBORN STREET, CHICAGO 10, ILLINOIS

THE MIDDLE EAR

Edited by

HEINRICH G. KOBRAK, M.D.

The contributors to this volume—John R. Lindsay, J. E. Fournier, Samuel Rosen, A. Wilska, H. Wullstein, Fritz Zoellner, and the late Dr. Kobrak himself—are known for their part in advancing surgical techniques for the cure of deafness. Their work represents the rejuvenation of otology as a specialty, and this book contains their latest findings in the treatment of both otosclerotic and non-otosclerotic deafness.

The new techniques—fenestration, mobilization, and tympanoplasty—necessitate extensive knowledge of anatomy, physiology, and bioacoustics. This the authors provide in the form of a solid background discussion of middle-ear morphology and function.

Six color plates and 240 black-and-white illustrations.

254 pages *$15.00*

At your bookstore or directly from the publisher:

The University of Chicago Press, 5750 Ellis Avenue, Chicago 37, Illinois

In Canada: The University of Toronto Press, Toronto, Ontario

Of interest to you and your patients

THE MENACE OF ALLERGIES

WHAT WE KNOW ABOUT ALLERGY
by Louis Tuft, 12 pages, 15 cents
HOUSE DUST ALLERGY
by Karl D. Figley, 8 pages, 15 cents
FOOD ALLERGY
by Samuel M. Feinberg, M.D., 6 pages, 10 cents
SKIN ALLERGY
by Samuel M. Feinberg, M.D., 6 pages, 10 cents
ASTHMA AND HAY FEVER
by Samuel M. Feinberg, M.D., 6 pages, 10 cents
RAGWEED AND HAY FEVER
by Oren C. Durham, 2 pages, 5 cents

AMERICAN MEDICAL ASSOCIATION
535 North Dearborn Street • Chicago 10 • Illinois

731/S

RHINOPLASTIC INSTRUMENTS
Designed By **DR. B. B. RISH**

Cat. No. 731/S RISH OSTEOTOME (Original Model) Stainless Steel, Overall Length 7" (18 cm.) $9.50 ea.

Designed for Intranasal Osteotomy.

Features. One instrument for both sides —Short, blunt, nonbreakable guard-guide, manually felt through skin follows exact osteotomy course. A Collar (ledge) over dorsal aspect which prevents slipping medially or laterally. Uniform thickness of 1 mm for 1½" (38 mm) from cutting edge to prevent separation of nasal fragments with premature low Fracture. Ref. Archives of Otolaryngology, Feb. 1953.

Cat. No. 717/S RISH CARTILAGE KNIFE, STAINLESS, $9.75 ea. Designed for "shaving" dorsal cartilage in Rhinoplasty.—Regardless of pressure exerted, it is impossible to remove more than a paper-thin portion of Cartilage.—

Ref. Archives of Otolaryngology, Oct. 1954.

Write for current Catalog No. 18.

B. J. FLORSHEIM
2067 Broadway, at 72nd. St.
New York 23, N. Y.

717/S

In acute and chronic

Sinusitis

By using the NICHOLS NASAL SYPHON, these cavities may be safely and successfully drained of mucopurulent material, thus affording prompt symptomatic relief. Used and prescribed for more than 41 years. Prove its effectiveness on your most troublesome cases. Just mail the coupon.

Nichols NASAL SYPHON

NICHOLS NASAL SYPHON, INC. MONTCLAIR, N. J.

Please send details of SPECIAL OFFER "S" without obligation

NAME..... M.D.

ADDRESS.....

CITY..... ZONE.....STATE.....

Chair—Upholstered, reclining ..$150.00

Cabinet—Stainless steel, eight drawers with or without complete transilluminator, cautery rheostat and transformer, waste container, air regulator, gauge, tubing and cutoff ..$115.00 to $195.00

Light—Telescopic .....$ 16.50

Cuspidor—With Suction .....$ 95.00

Stool—Upholstered, with easy running ball-bearing casters ..$ 20.00

Light Shield .....$ 5.00

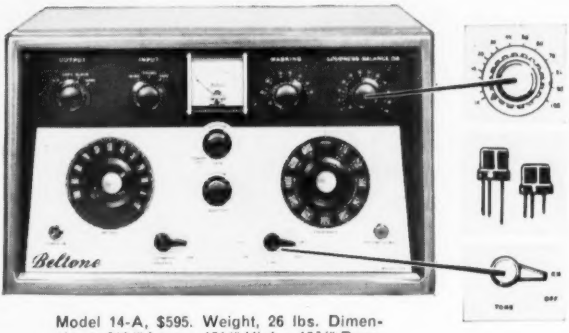
All equipment shown in the above reproduction (with spray bottles, stainless steel cotton jars, ready to be attached to your compressed air system, $531.50 F.O.B. Factory.

Catalogue sent on request

1901-1905 Beverly Blvd. SMR Los Angeles 57, Calif.

Doctor:

Beltone's new Diagnostic Audiometer-Model 14-A brings you these exclusive features! ...



Model 14-A, $595. Weight, 26 lbs. Dimensions: 21½" Long x 13¼" High x 13¾" Deep.

EXCLUSIVE! *Simplified loudness balance control permits Fowler alternate loudness balance binaural recruitment tests by merely pressing a switch. Automatically puts tones of different intensities alternately into each ear. Eliminates calculations!*

EXCLUSIVE! *Transistorized speech circuit greatly reduces hum interference in both recorded and live speech tests.*

EXCLUSIVE! *Extremely fast acting tone switch for precise appraisal of patient reaction. Complete rise and decay of tone takes place in only .1 second.*

MODEL 14-A PERFORMS THESE SPEECH AND PURE TONE HEARING TESTS:

- **Pure tone air conduction**
- **Pure tone air conduction with masking**
- **Pure tone bone conduction**
- **Pure tone bone conduction with masking**
- **Alternate loudness balance, binaural**
- **Equal loudness contours, monaural**
- **Shifting voice**
- **Lombard**
- **Doerfler–Stewart**
- **Stenger (Stenger box–$10 as an accessory)**
- **Speech–live voice**
- **Speech–recorded voice (tape or phono)**
- **Audiometer Weber**
- **Above threshold audiogram**
- **Masking audiogram**
- **Pitch difference limen (with accessory)**

The Model 14-A Audiometer, designed especially for diagnostic use by physicians, hospitals and clinics, offers you new advantages in fast, extremely accurate hearing evaluations. Leading otologists and members of the medical profession are enthusiastic about this outstanding instrument. For complete information on the Model 14-A Diagnostic Audiometer, have your nurse mail us the coupon and we will rush you a fully illustrated, factual brochure. No obligation, of course.

MAIL COUPON FOR YOUR FREE BROCHURE

Beltone Hearing Aid Company, Dept. 9-209
2900 W. 36th St., Chicago 32, Illinois

Please send me your free brochure describing the Model 14-A Diagnostic Audiometer. I understand this does not obligate me in any way.

NAME _____

ADDRESS _____

TOWN _____ ZONE _____ STATE _____

☐ Please check here if you wish information on Beltone's Model 15-A Two Channel Clinical Audiometer or the portable line.

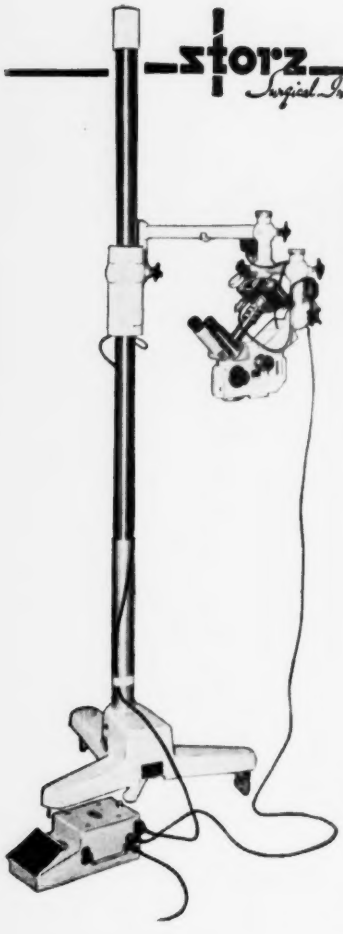
Beltone®

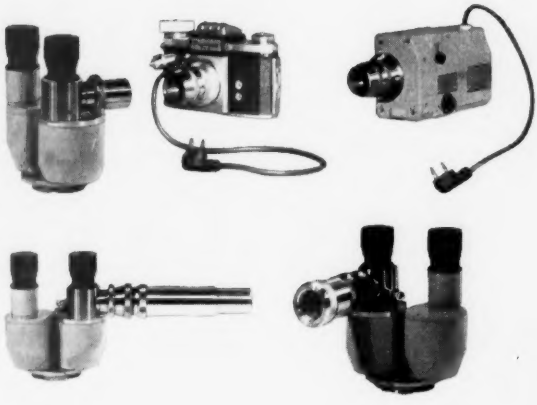
AUDIOMETERS

Produced by the Beltone Hearing Aid Company
2900 West 36th Street, Chicago 32, Illinois

world's largest exclusive manufacturers of precision audiometers and transistor hearing aids

storz
Surgical Instruments





OPERATING MICROSCOPE CAMERA *(SHEA)*

With this equipment it is possible to photograph while operating, without disturbing the operator's vision. Either 35mm slides or 16mm motion pictures may be made by simply interchanging cameras on the binocular take-off tube. This take-off tube may also be utilized to use the auxiliary eyepiece.

N-987 Fig. A Binocular Take-Off. This adapter can be fitted to any Zeiss binocular head. Customer must supply binocular head.* Both 35mm and 16mm cameras fasten directly to take-off tube with collet ring lock. Focus ring need not be changed $495.00

N-987 Fig. B Focuser. Screws directly to the camera collet fastener. Gives good view of image. May be used by an observer 18.00

N-987 Fig. C Auxiliary Eyepiece. For assistant or observer. Places observer at right angles to surgeon. Complete with 12X lens 125.00

N-987 Fig. D 35mm Camera, Solenoid Operated. Removable Teflon cap for film advance knob may be autoclaved .210.00

N-987 Fig. E 16mm Motor Driven Motion Picture Camera. Best results with Super Anscochrome color film 515.00

N-987 Fig. F Trans-Switch. Operating microscope cord plugs into trans-switch. Button switch provides 6 volts to microscope. Foot-pedal switch simultaneously increases light brilliance and activates either camera solenoid or movie camera motor 177.50

N-987 Fig. G Fitted Carrying Case. (Not illustrated) 50.00

Set Complete..... $1590.50

*(Binocular head supplied, with 12.5X lens, for an additional $275.00).

STORZ INSTRUMENT COMPANY

4570 Audubon Avenue, St. Louis 10, Mo.

New York Showroom, 809 Lexington